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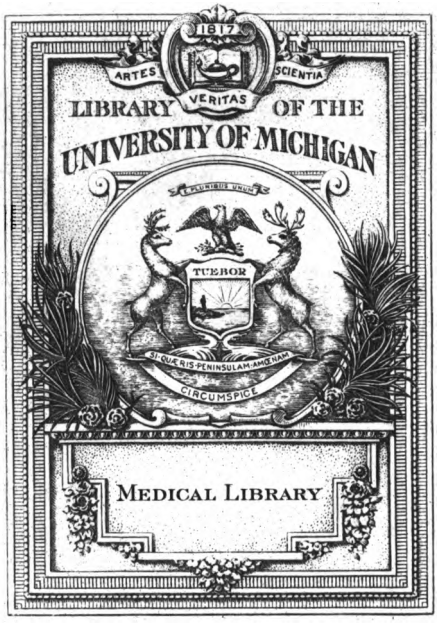

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TRANSACTIONS OF THE

SECTION ON

Nervous and Mental Diseases

of the
American Medical Association
at the Seventy-First Annual
Session, held at New Orleans,
La., April 26 to 30, 1920

AMERICAN MEDICAL ASSOCIATION PRESS
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LIST OF OFFICERS

This Section has been in existence under its present name since 1900. The branches of the practice of medicine which comprise its province were classified with some other subjects in the early days, as is shown by the following section names. The list of officers has been compiled from the published records, which are not complete—for example, the city is not given in some cases. It will be appreciated if any additional information is brought to the attention of the Secretary of the American Medical Association.

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1869 Records wanting.

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1871 Records wanting.

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1897 Chairman, C. H. Hughes, St. Louis.
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1898 Chairman, F. Peterson, New York.
Secretary, H. T. Patrick, Chicago.

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Vice Chairman, Walter Timme, New York.
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PROCEEDINGS OF THE SECTION

WEDNESDAY, APRIL 28—MORNING

The meeting was called to order by the vice chairman, Dr. Arthur S. Hamilton, Minneapolis.

On motion by Dr. C. R. Woodson, St. Joseph, Mo., carried, Drs. Hugh T. Patrick, Chicago; Karl A. Menninger, Topeka, Kan., and Edward L. Hunt, New York, were appointed as a committee to draft suitable resolutions on the death of Dr. Elmer E. Southard, and report at a subsequent session.

Drs. Ross Moore, Los Angeles; William A. Jones, Minneapolis, and Roy M. Van Wart, New Orleans, were appointed to act on the executive committee.

Dr. Arthur S. Hamilton, Minneapolis, read the chairman's address, entitled "Some Changes in the Nervous System in Pernicious Anemia, Especially Sensory Changes."

Dr. Edmund Jacobson, Chicago, read a paper on "Reduction of Nervous Irritability and Excitement by Progressive Relaxation." Discussed by Drs. Tom A. Williams, Washington, D. C.; Hyman Climenko, New York, and Edmund Jacobson, Chicago.

Dr. Gustave Roussy, professor of medicine, University of Paris, France, was introduced by Dr. Hugh T. Patrick, Chicago. In response to the cordial welcome extended, Professor Roussy expressed his pleasure in being present on this occasion and in meeting with the great American Medical Association, whose name is so well known in France. He expressed pleasure concerning the close association of American and French neurologists which has been brought about through the war, and voiced the desire that the relations of French and American institutions might continue—that the French might come to American colleges and learn of us, and we go to their educational institutions and learn of them, and so cultivate that broad spirit which makes for the best relations between nations.

Dr. Karl A. Menninger, Topeka, Kan., read a paper on "Influenza and Feeblemindedness." Discussed by Drs. Andrew L. Skoog, Kansas City, Mo.; Hyman Climenko, New York; Oscar J. Raeder, Boston; Tom A. Williams, Washington, D. C.; C. F. Neu, Indianapolis, and Karl A. Menninger, Topeka, Kan.

Dr. Sanger Brown, Chicago, read a paper on "Outline of a Scheme for Writing the Natural History of Syphilis." Dis-

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cussed by Drs. C. R. Woodson, St. Joseph, Mo., and Sanger Brown, Chicago. In concluding his paper, Dr. Brown introduced and moved the adoption of the subjoined preambles and resolutions:

WHEREAS, The deleterious effects of syphilis on the mortality and morbidity of the human race are so prevalent and so severe as to challenge the most serious attention of the medical profession; and

WHEREAS, In the scientific study of any disease, knowledge of its natural history is a matter of cardinal importance; and

WHEREAS, Owing to the protracted course of syphilis, a continuous, complete clinical record of a given case can be secured only through the services of several successive medical observers; and

WHEREAS, It is highly desirable that a sufficient number of completed histories be accumulated and preserved and made easily accessible to students; and

WHEREAS, For the successful accomplishment of the purpose set forth above, the interest and cooperation of the best elements of our profession as represented in the membership of the American Medical Association are necessary; therefore, be it

Resolved, That the Section on Nervous and Mental Diseases of the American Medical Association recognizes the importance of ascertaining the natural history of syphilis and of making the history accessible for the information of students of medicine; and be it further

Resolved, That the Section on Nervous and Mental Diseases of the American Medical Association respectfully requests the trustees of the American Medical Association to appoint a committee from the sections most immediately concerned whose duty it should be to devise practical means and methods of accomplishing the foregoing specified purpose; and be it further

Resolved, That the representatives of the section in the House of Delegates be requested to present these preambles and resolutions to the House of Delegates and to ask its endorsement.

See minutes of the House of Delegates.

Dr. Hugh T. Patrick, Chicago, moved the adoption of the resolutions. Motion seconded by Dr. W. S. Lindsay, Topeka, Kan., and unanimously carried.

The following members were appointed to act as the nominating committee: Drs. George A. Moleen, Denver; Frank R. Fry, St. Louis, and Walter Timme, New York.

Dr. William A. Jones, Minneapolis, read a paper on "Discussion of Therapeutic Agents in Chronic Nervous Diseases." Discussed by Drs. Frank R. Fry, St. Louis; Ross Moore, Los Angeles; C. R. Woodson, St. Joseph, Mo.; Hyman Climenko, New York; W. T. Williamson, Portland, Ore., and William A. Jones, Minneapolis.

Dr. Frank R. Fry, St. Louis, read a paper on "Congenital Facial Paralysis: Two Additional Cases." No discussion.

THURSDAY, APRIL 29—MORNING

The section was called to order by the chairman at 9:30.

Dr. Julius Grinker, Chicago, read a paper on "Experiences with Luminal in Epilepsy." Discussed by Drs. George A. Moleen, Denver; Marvin L. Graves, Galveston, Texas; Andrew L. Skoog, Kansas City, Mo.; David S. Booth, St.

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Louis; E. Bates Block, Atlanta, Ga.; F. B. Wynn, Indianapolis; Roy M. Van Wart, New Orleans, and Julius Grinker, Chicago.

Dr. Hugh T. Patrick, Chicago, presented the report of the committee appointed to draft resolutions on the death of Dr. Elmer E. Southard, as follows:

WHEREAS, Dr. Elmer E. Southard, who one year ago was elected chairman of the Section on Nervous and Mental Diseases of the American Medical Association and should have presided at this session, has been removed by death: and

WHEREAS, Dr. Southard was a trained pathologist, a gifted neurologist, an able psychiatrist, an author of distinction, a brilliant teacher, a genial companion and a loyal friend; therefore be it

Resolved, That in the death of Dr. Southard the medical profession and especially the departments of neurology and psychiatry have suffered an irreparable loss: and be it further

Resolved, That the members of this section extend to the family of Dr. Southard their profound sympathy.

On motion duly seconded and carried, the foregoing preambles and resolutions were adopted by the section.

Dr. Oscar J. Raeder, Boston, read a paper on "Endocrine Imbalance in the Feebleminded." Discussed by Drs. Tom A. Williams, Washington, D. C.; Gustave Roussy, Paris, France; Walter Timme, New York; M. A. Bliss, St. Louis; Andrew L. Skoog, Kansas City; Karl A. Menninger, Topeka, Kan., and Oscar J. Raeder, Boston.

Dr. I. Leon Meyers, Chicago, read a paper on "The Physiologic Significance of the Babinski Toe Response." Discussed by Drs. Andrew L. Skoog, Kansas City; Tom A. Williams, Washington, D. C.; Gustave Roussy, Paris, France; M. A. Bliss, St. Louis, and I. Leon Meyers, Chicago.

FRIDAY, APRIL 30—MORNING

The meeting was called to order at 9:30 by the chairman.

The following officers were elected for the ensuing year: chairman, Dr. Arthur S. Hamilton, Minneapolis; vice chairman, Dr. Walter Timme, New York; secretary, Dr. Charles W. Hitchcock, Detroit (continued), delegate, Dr. Hugh T. Patrick, Chicago.

Drs. Isador Abrahamson and Hyman Climenko, New York, presented a paper on "Symptomatology of Spinal Cord Tumors with Illustrative Cases." Discussed by Drs. Julius Grinker, Chicago; Alfred W. Adson, Rochester, Minn.; A. L. Skoog, Kansas City, Mo.; Karl A. Menninger, Topeka, Kan.; M. A. Bliss, St. Louis; George A. Moleen, Denver; Tom A. Williams, Washington, D. C., and Hyman Climenko, New York.

Dr. Tom A. Williams, Washington, D. C., read a paper on "The Causes of Emotivity and Their Management." Discussed by Drs. Julius Grinker, Chicago; David S. Booth, St.

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Louis; Hyman Climenko, New York; Karl A. Menninger, Topeka, Kan.; C. F. Neu, Indianapolis; Ross Moore, Los Angeles; G. H. Benton, Miami, Fla.; Gustave Roussy, Paris, France; George A. Moleen, Denver, and Tom A. Williams, Washington, D. C.

Dr. A. L. Skoog, Kansas City, Mo., read a paper on "Measles: Brain Complications." Discussed by Drs. Karl A. Menninger, Topeka, Kan., and A. L. Skoog, Kansas City.

Dr. E. Bates Block, Atlanta, Ga., read a paper on "The Relation of Worms to Epilepsy." Discussed by Drs. Julius Grinker, Chicago; David S. Booth, St. Louis; Hyman Climenko, New York; Martin L. Graves, Galveston, Texas; Tom A. Williams, Washington, D. C., and E. Bates Block, Atlanta.

Dr. Edward Livingston Hunt, New York, read a paper on "Encephalitis Lethargica." Discussed by Drs. Frank R. Fry, St. Louis; Julius Grinker, Chicago; Tom A. Williams, Washington, D. C.; Ross Moore, Los Angeles; Hyman Climenko, New York; George A. Moleen, Denver; Oscar J. Raeder, Boston; A. L. Skoog, Kansas City, Mo.; Roy M. Van Wart, New Orleans; Albert Woldert, Tyler, Tenn., and David S. Booth, St. Louis.

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REDUCTION OF NERVOUS IRRITABILITY AND EXCITEMENT BY PROGRESSIVE RELAXATION

EDMUND JACOBSON, A.M., PH.D., M.D.
Associate Attending Physician, Michael Reese Hospital
CHICAGO

It is generally admitted that there is a lack of adequate treatment for what is commonly called nervousness. Often the patient is told that there is nothing the matter with him—that he should go home and forget himself. If there is an apparent pathologic condition, as in postoperative cases, little is done to quiet the nervous system, although the suffering here may outlast the organic. It is forgotten that relief from functional ailment may aid the organism in its struggle against other disorders. In consequence of repeated futile efforts to get relief from legitimate sources, the public naturally turns to Christian science, chiropractic and other charlatans or healers.

The medical public has generally considered the Weir-Mitchell rest-cure a step in the right direction. Yet there seems to be a strange neglect on the part of that discoverer and his followers of the underlying physiology; the method seems to have been used in an empiric way, without penetration to the cause of its success; for, evidently, the aim is to achieve habitual relaxation, yet this word seldom occurs in his works or in those of most other neurologists. Accordingly, it seems logical to search in the direction of physiologic relaxation for a more direct and efficient means of bringing quiet to the nervous system.

This point was indirectly suggested by experiments begun at Harvard University in 1908, later con-

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tinued at Cornell, and recently at the University of Chicago. The significance of relaxation was noted, and observations were made of the behavior of individuals as they progressed toward mental and emotional passivity. In this way we obtained objective signs of progress toward relaxation which we could compare with the individual's own reports.

It seems evident, when attention is called to the matter, that excitement and irritability of the cerebrospinal nervous system will show itself in the form of motor symptoms or signs. Striated muscles will be overactive, and speech or other functions will reveal the disturbance. There may be observed: wrinkling of the forehead; frowning; eyelids widely opened or tensely squinting; frequent winking; eyes moving restlessly or prolongedly staring; tightness of the lips, perhaps with a downward curve at the angles of the mouth; face muscles tense or quivering; speech rapid or broken, with variable or high pitch; frequent swallowing; the head held stiff or moving restlessly; thoracic or abdominal breathing, rapid or irregular and violent; the limbs and trunk tense or shifting restlessly from time to time. This list, of course, is far from exhaustive. Overactivity of a group of striated muscles may show itself in quivering and tremor, in restless, jerky movements, or in a tensely fixed position. The observer may interpret what he sees and hears, and say that the patient appears anxious or worried or fearful or mentally overactive. In everyday life we make such interpretations unawares, without stopping to note and classify how we do so. Accordingly, we may fail to note fine signs of nervous or muscular tenseness, such as restless movements of perhaps only a finger, or slight shifting of the eyes. It would seem that excitement, even if mild and chronic, cannot readily be concealed from an observer who makes free use of his eyes and ears, to say nothing of tests of reflexes and passive movements and palpation of tenseness of muscles.

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Symptoms and signs of overactivity of the autonomic system may accompany those of the cerebrospinal system, but are familiar to all and need not here be described. It is rather the latter that have been neglected.

Overactivity or increased tonus of the central nervous system, as shown in tenseness or exaggerated or excessive movement of striated muscles, is subject to voluntary control. Every individual uses this natural function when he goes to rest. It would seem strange, therefore, if it could not be specially cultivated to counteract an excess of activity and bring quiet to the nervous system. Such is the aim of the present method. As may be readily noted, the neurotic individual has partly lost the natural habit or ability to relax. Usually he does not know when to relax, and cannot judge accurately whether he is relaxed. These things must be cultivated. Accordingly, it is usually futile to tell the sufferer to relax or to have him take exercises to this end in gymnasiums. Furthermore, relaxation as heretofore understood is not adequate as a clinical method. Following previous standards, a patient may be apparently "relaxed" in bed for hours or days, yet be worried, fearful or otherwise excited. In this way the individual has been wrongly considered relaxed, overlooking voluntary or local reflex activities shown by such slight marks as wrinkling of the forehead, tenseness about the eyes, and restless shifting of a limb or even a finger. The present method notes such signs in attempting to bring about nervous and mental quiet.

According to my experience, if the patient is shown how to relax the voluntary system, there later tends to follow a similar quiescence of the autonomic apparatus. Emotions tend to subside as he relaxes. To be sure, there may be a vicious circle: autonomic overactivity seems to stir up local or general cerebrospinal reflexes, and these again autonomic reflexes. The one system must become quiet before the other system can become

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quiet. So in certain chronic cases, relaxation becomes a gradual progress, a matter of habit formation, wherein the presence of pain or disordered internal secretion or other organic disturbance may partially or completely block the way.

In this connection it seems pertinent to inquire, What is the relation of nervous and muscular relaxation to the quieting of mental excitement? We are reminded that the relaxation of natural sleep as well as that produced by narcotics and anesthetics is accompanied step by step by mental quiet. Furthermore, the question apparently can be answered by taking into account observations familiar to experimental psychologists. For example, Meumann [1] observes, "The occurrence of tenseness (Spannungen) in the muscles of the body accompanies concentration of the attention." He refers to the muscles of the sense organs, eyes, face, limbs, neck, jaws and others. In certain of the experiments mentioned, I found that as such motor signs subside, the individual's mental activity or attention diminishes.[2] If they subside sufficiently, he naturally falls asleep. Similarly, according to my clinical experience in about twenty-five cases, as motor signs subside the individual becomes less excited and irritable.

In calming mental excitement by physical relaxation, two difficulties must be overcome: 1. The patient will not be able to relax at first in cases in which it is not possible to remove the stimulus to the mental irritation. The unfortunate person wishes to worry and fret, wishes to find a solution of his problems, and in so doing becomes tense, as may be externally observed. In the presence of grave troubles, as is well known, it does little good to tell the afflicted one to stop thinking, to stop fretting. On the other hand, he may be more readily shown how to bring about this mental control through physical relaxation. 2. According to

1. Meumann: Vorlesungen, Leipzig, 1907.
2. Jacobson, Edmund: Further Experiments on the Inhibition of Sensations, Am. J. Psychol., July, 1912.

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my observations, apparent contraction of the muscles of the eyes, lips and facial expression play an important rôle in mental irritability and excitement. Some patients have called my attention also to those at the back of the head and neck. It is of course by expression in these parts, by varying tenseness and relaxation, that persons are accustomed to convey their meanings, so that in the face of the individual one may read his state of mind. The muscles here involved are small, and the sense of contraction is relatively faint. Accordingly, it sometimes becomes a delicate matter to make the patient recognize tenseness in these small muscles. He may lie on a couch with limbs, trunk and neck well relaxed, but with slight wrinkling of the forehead or quivering of the closed eyelids indicating the presence of mental activity. Special drill may have to be given at this point in chronic insomnia.

It is, of course, true that the average person does not know when he is tense. Psychologic experiments have made clear how sensations of tenseness or contraction constitute some of the very essence of the process of thinking, but this does not mean that the thinker is aware of the sensations as such. In the present method the patient is made aware of sensations of contraction, and later may observe such contractions taking place while he thinks or is excited. He may note how tense he becomes; and his attention, thus drawn to the point, leads him to relax; or again, when he becomes tense, the habits he has newly acquired may lead him automatically to repose.

When the unpracticed person lies as quietly as he can on a couch, it is found from external signs and tests that the relaxation is incomplete. There remains over what may conveniently be called *residual tension*. The same may also be inwardly observed, of course, through the kinesthetic sense. Many years of observation on myself have suggested that insomnia is always accompanied by a sense of residual tension and can

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always be overcome when one successfully ceases to contract the parts in this fine degree. Residual tension, accordingly, appears to be a voluntary fine contraction. On the other hand, of course, it may be reflexingly stimulated, as by pain; yet relaxation may be of service even here.

Doing away with residual tension is, then, the essential point of the present method. This cannot happen in a moment, even in the practiced person. The tension only gradually disappears; it may take fifteen minutes progressively to relax a single part, such as the right arm. The desired relaxation begins only at the moment when the patient might appear to an inexperienced observer to be very well relaxed.

The present method consists of voluntary continued reduction of contraction or tonus of muscle groups and of motor or associated portions of the nervous system. The relaxation is progressive in three respects: 1. The patient relaxes a group, for instance, the biceps-brachial of the right arm, further and further each minute. 2. He learns consecutively to relax the principal muscle groups of his body. With each new group he simultaneously relaxes such parts as have previously received practice. 3. As he practices from day to day, he progresses toward a habit of repose. He tends toward a state in which quiet is automatically maintained. In contrast with this, the individual who indulges in unrestrained excitement renders himself liable to further increase of excitement. This is the familiar physiologic law of augmentation.

Before learning to relax any muscle group, the patient is first made to contract it, with closed eyes noting the sensation from the contraction.[3] This accomplishes two things: 1. It enables him to recognize the location of tenseness during moments of excitement and so specifically to relax the part. 2. It brings home to him that the tenseness which takes

3. For a discussion of the muscular sense, compare Woodworth: Le Mouvement, 1903, or the works of C. S. Sherrington.

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place in nervous or mental excitement is a part of his own voluntary activity and therefore can be relaxed.

It has not seemed best to me to secure the patient's cooperation by suggestions and promises. Rather, his symptoms of overactivity are pointed out, if he has not already noticed them himself, and he is shown how to do away with them in an independent manner. In this way improvement takes place *pari passu* with progressive relaxation and not by dint of suggestion. To be sure, the present method may be combined with suggestion or other psychotherapy, with drugs, surgery, or what may be required.

Instructions may be carefully but colloquially worded. The patient is directed to relax the part or parts "further and further each minute." Movements soon come to be recognized as interfering with relaxation, but he is not to make any effort to hold himself still. No task is set, but rather he is "not to bother to do anything with his arm" or "not to bother to look at anything." For example, after most of the muscle groups have been covered, the instruction delivered quietly and with many pauses may be: "Now relax your right arm in front and in back, above and below! Just let it go further and further every minute! Let the left arm go! and the right leg; the calf and in front; and the thigh—in back and in front—and the hip behind and in front! Let the left leg go also! Don't bother to do anything at all with your arms and your legs! Now let the back become as limp as a rag! And the abdomen and the chest: just let your breathing go of itself! And your neck! Let the head go completely! And the forehead! Don't bother to wrinkle your forehead! The eyes! Let them go completely Don't bother to look anywhere! The tongue and lips and face! Just let the throat go entirely!" Of course, instructions vary according to the progress of the patient and the location of tenseness. The practiced person aims to note the location of tenseness

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himself and to do away with it. However, he is not to concentrate his attention on his muscles, since this requires effort, which is to be avoided. He is just to let go and not even to "bother" if he fails to relax. All effort, including effort to relax, is, of course, to be avoided.

Relaxation may be called "general" when the whole body is on a couch or bed. Perfect general relaxation does not take place. As repose progresses, the individual tends to fall asleep. Occasionally some persons, even insomniacs, do so in the very first hour of practice. Individuals differ, some being able to relax better in the absence of the physician. Needless to say, no suggestions to sleep in the technical sense are given, and no *rapport* is established.

Relaxation may be called "relative" when the patient is active, provided only such muscles are in use as are needed. For example, a pianist may be excited while he plays, as shown by a frown, frequent restless movements of the head, and needless shifts of position; or he may play in a relaxed manner. After acquiring general relaxation, the patient sits up with eyes closed, and is practiced in relaxing one muscle group after another, as before. Those muscles engaged in maintaining posture need not be markedly relaxed. Thereafter the patient may open his eyes and engage in conversation while the physician watches closely for signs of undue tension, drawing notice to these when they appear. Again, the patient may be observed while at some occupation and shown how to keep relatively relaxed during his activities. In this way he may avoid irritability and excitement, forestalling periods of nervousness even while he continues to be active.

What is the relation of suggestion to the present method? The use of suggestion is to be carefully avoided. One need not tell the patient that he will benefit. It suffices to show him how to relax. The student of the subject will know that relaxation does

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not depend on suggestion, but rather is a fundamental or elementary physiologic process. It may be found in low grade forms of life, such as "nerve-muscle preparations" in the laboratory, where no question of suggestion can arise. Of course, in the human organism neuro-muscular relaxation may be brought about under appropriate conditions by means of suggestion, as well as by change of scene, good fortune, sedatives, baths and psychotherapy and other means. However, the present method rests not on such means, but simply on cultivating the natural ability of the individual to relax his parts.

The relation of the present technic to what is popularly called relaxation and to the methods of a few physicians who have recommended muscular relaxation was discussed in a previous paper. It is important to avoid confusing what I have called progressive relaxation with means previously taken to get the patient to relax; for hitherto residual tension has been overlooked, although doing away with this is the sine qua non of thorough and successful treatment. Apparently it is possible for an individual to be inwardly excited — worried, enraged, anxious or restless — unless residual tension has been overcome.

Treatments vary in duration according to the condition of the patient and the time at the disposal of the physician. In my own practice they have taken from one half to two hours, one case requiring as few as two treatments, another as many as ninety. However, for general practice the periods doubtless may be shortened to twenty or thirty minutes.

The method may be abridged in any one or more of four ways: 1. The patient may be shown how to recognize tenseness serially in the principal muscle groups of the entire body and how to relax them, in a single session or two. This may suffice for quick results in acute cases. However, in stubborn cases it has been my practice to devote the first session to one arm or a

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portion thereof. 2. Cultivation of the ability to recognize tenseness may be omitted. 3. In its simplest form, the method consists of directing the patient to relax whatever parts appear to the physician to be tense, without previous training as to muscle groups, and without noting his sensations.

A simple way to shorten the procedure is by selecting only a few muscle groups for practice. For instance, only the biceps-brachial group may be used in the arm, and only the calf muscles in the leg. In this way the patient proceeds at once to relax his whole arm when he begins to relax the flexors of the forearm. The principle here is that progressive relaxation of one part tends to bring with it a similar condition in other parts.

The present method is not offered as a panacea for mental disorders. Rather, if necessary, it may be used in conjunction with any other form of treatment. The cases presented herewith are offered as illustrations rather than as proofs of the efficacy of the method. Any one who has cultivated the habit of observing tenseness of his fellow men in their daily pursuits, and who has noted the exaggerated tenseness of the neurotic individual, presumably will see the importance of the principle of relaxation. Recurrences may be anticipated in some cases. Relaxation for nervousness apparently is like diet or hygienic measures in gastro-intestinal disorders. The permanence of the results will depend on the care with which the physician gives his instructions, and the zeal with which the patient follows them. It is hoped that this method may be adapted to the use of the general practitioner. However, time and patience are required; the physician himself must know how to relax before he can show the patient. The principle is simple and obvious, but the technic involves many points for successful operation.[4]

4. For further details, see also Jacobson, Edmund, Use of Relaxation in Hypertensive States, New York M. J. **111**: 419 (March 6) 1920.

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REPORT OF CASES

CASE 1.—Mrs. Z., an excitable, restless and talkative society woman, who feared the approach of the menopause, was subject to occasional pyrosis, pruritus ani, frequent urination and to seasonal spells of hay-fever and bronchitis. General physical examination was practically negative, including blood, urine and roentgen-ray examination of chest. She was somewhat obese. She gave a history of a homosexual strain and anomalous sexual difficulties dating back to childhood.

Treatment was begun with psycho-analysis. This seemed to assist toward better mental adjustment, but left her excitable and overactive as before. A more restful attitude and a more quiet deportment have been secured with progressive relaxation, in weekly or biweekly sessions for six months.

CASE 2.—A "compulsion neurosis" in a woman, aged 31. An excavated ulcer near the middle of the right sternocleidomastoid muscle, about 3 by 1.5 cm. in size, was caused by picking at it with her fingers. A few small scars remained on the face from previous self-mutilations. Torn between two loves, one for her husband from whom she was perhaps to part, she was wretched with indecision. As those mental causes of disturbance could not be removed, she was shown how to relax. Her attention was called to the physical manifestations of her excitement and worry, including the nervous movements of her hands towards her neck. She did not succeed in acquiring relaxation of the eyes and face sufficient for marked mental repose, but after a month's treatment she felt that her equilibrium was restored and she returned to her work and domestic problems with no further medical assistance for many months. After relaxation was begun the ulcer rapidly healed, with the aid of hot boric acid dressings.

CASE 3.—This illustrates the use of progressive relaxation in addition to drugs in organic disorders. A woman, aged 43, complained of spells of fearful palpitation accompanied by tremor, chilliness, nausea and fear of death, as well as "trembling" in the epigastric region. These spells began three years ago when she was jaundiced for a whole year. Salpingectomy in 1912 followed chronic suppuration. Examination disclosed, principally, loss of weight, hemorrhoids, and purulent fissure in ano. Wassermann test ++. There was no other marked abnormality. The patient was of course reassured that there was no organic affection of her heart. She had heard this from previous physicians. She received three half-hour treatments per week of progressive relaxation. Practically from the outset, with few exceptions, she became able to avert the spells and to relax away the symptoms of anxiety. The case is still under treatment. In this manner it has been possible to proceed in an unimpeded way with mercury and iodids.

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CASE 4.—A girl, aged 26, had insomnia and restlessness, with symptoms of early hyperthyroidism, daily spells of nausea for many months. There was moderate general enlargement of thyroid, a somewhat rapid pulse, afternoon rise of temperature, flushing of skin, positive Goetsch test and alimentary hyperglycemia following the glucose test meal. The first basal metabolic test was 18 above normal. The gastric content lacked free hydrochloric acid. Roentgen-ray examinations of lungs, stomach and intestines were negative, as were also the Wassermann and tuberculin tests. The uterus was in retroversion.

Foci of infection were removed from the teeth. A prolonged rest cure was given, with early use of roentgen rays on the thyroid, later quinin hydrobromid, sodium phosphate and empirical remedies for the nausea.

Before I saw her, the patient had received bromids and other sedatives for insomnia and restlessness. She complained that these drugs failed to quiet her at night, and depressed and confused her by day. Accordingly, they were stopped, and the insomnia was relieved at once after one or two treatments with progressive relaxation. She soon regained her former mental buoyancy. Treatments have been continued toward perfecting her ability to relax.

CASE 5.—This illustrates the obvious application of progressive relaxation to the treatment of tics. The patient, a married woman, aged 31, had a spasmodic, rotary movement of the head at irregular intervals of years' standing. It was apparent that she was generally tense. After she had learned to relax her limbs, she failed to return for further treatments, owing to illness in her family. When last seen she reported that with daily practice at relaxation the movement had been largely absent. This perhaps indicates how relaxation of one part—here the limbs—may bring with it relaxation of another part—here the muscles that move the head.

30 North Michigan Avenue.

ABSTRACT OF DISCUSSION

DR. TOM A. WILLIAMS, Washington, D. C.: The principles outlined by the author have been used in Switzerland for about twenty years. About fifteen years ago, the method was adopted by Crighton Miller in London, who later wrote a paper about it. I tried the method ten or twelve years ago, but found that either I am not suited to using that elaborate method, or it is not necessary for most patients. The secret of the failure to cure tics by exercise has seemed to me to be too great insistence on the method advocated by Meige, and that the real secret of getting well of tic is to teach the method of self-control rather in the way Dr. Jacobson has advocated. Of course, we know very well that certain cults

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in this country have for many years pursued methods of tranquilizing in particular method known as "power through repose," in which the principle is practically the same as that employed by Dr. Jacobson except that he addresses himself to the specific motor excitation. By so doing one can entirely remove a tic, more especially an abdominal tic, in one sitting. I have done it frequently, having such patients relax thoroughly. They recover entirely after one has taught them how to obtain control of a series of movements which are usually automatic. It is more difficult in tic of the face or neck. The reason seems to be that the patient has exhausted many experiments in order to control certain muscles, and when you try to teach a new method you are hindered by the automatic movements which the patient has learned to make in order to evade the tics. The point of teaching the patient to relax the arm or leg before directing specific attention to the neck or face is, I think, a most valuable one. The technic Dr. Jacobson has given will bring the attention of many of us to a most important part of therapeutics.

Dr. H. Climenko, New York: Dr. Maloney has made use of relaxation in the treatment of locomotor ataxia. He uses this method mainly to overcome fear, which he believes is a strong factor in syphilitic patients. This method of treatment is now being used successfully in Dr. Bernard Sachs' service at the Mt. Sinai Hospital. I have resorted to relaxation in the treatment of neurotic patients both at the clinic and in private practice. While I had some success with some patients, in many no results were obtained. I believe that the degree of improvement is not altogether in proportion to the ability of the patient to relax. Suggestion plays a great rôle in this form of treatment.

Dr. Edmund Jacobson, Chicago: In the past, workers in this field have not regarded the element to which I tried to call particular attention—that of residual tension. It is disregard for that very fine element of tenseness that remains after the individual is ordinarily called relaxed which has prevented results. I believe that this will also explain the poor results referred to by Dr. Climenko. Unless you consider the residual tension you have not the essential point of this method. There are other ways of bringing about relaxation, but they are not so well adapted for clinical use. It is only when you take into consideration residual tension as shown by slight signs, and overcome it, that the patient will relax, and not merely in an ordinary gymnastic sense—the sense in which the term has been used by physicians in the past. In regard to habit spasms, one does not have to relax the individual part in order to get reduction of the habit spasm.

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INFLUENZA AND HYPOPHRENIA

THE INTERRELATION OF AN ACUTE EPIDEMIC INFECTION AND A CHRONIC ENDEMIC (BRAIN) AFFECTION

KARL A. MENNINGER, M.D.
TOPEKA, KAN.

Influenza seems to have had certain neuropsychiatric effects. It is these effects which are to us as physicians most important from the pragmatic standpoint, rather than any hypothetic propositions of cause. These concrete effects we may approach as tangible globar material, and it is the essence of pragmatism that to capitalize this concreteness will in the end yield results nearer truth than the spinning, however deft, of tenuous inductive hypotheses. Hughlings Jackson put it that "the study of the thing caused must precede the study of the cause of the thing."

The whole subject of mental defect stands in a fair way to illustrate the value of the pragmatic point of view. After hundreds of years of desultory search for *causes,* we have within the last few decades turned our attention to the *effects* of feeblemindedness, with the result that more advancement has been made in that short space of time than in all the preceding centuries; for the fact remains that however important the cause (and who will deny this?) it is in general a thing difficultly accessible; yet, in the pursuit of it, the obvious data of effects, from which so much can be learned, have been ignored.

Influenza, as I would demonstrate, had certain definite effects on hypophrenia, or feeblemindedness. The importance of this fact, and these data, in arriving at a correct conception of the nature of feeblemindedness must be obvious. Yet that such effects of

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influenza have ever occurred appears to be a naive observation. I mean simply that the literature is almost barren of any discussion of the matter. The whole problem of the effects of acute infections on the brain and its diseases is a strangely neglected one, and particularly is this true of feeblemindedness. It is only from the standpoint of alleged cause that infectious disease is discussed by writers on feeblemindedness, and even as such it is not even mentioned by many of them, and by others only casually.

Thus, to illustrate, Tredgold[1] in his well-known treatise on mental defect, devotes only eighteen lines (1908 edition) of the nearly 400 pages to the discussion of infections as causes of hypophrenia. Ireland[2] devotes a chapter to "Inflammatory Idiocy," but it is not very illuminating, and infectious disease is not mentioned in the chapter on "Causes of Idiocy." Bourneville[3] was apparently more interested in the structural alterations wrought than in the modus operandi by which they were wrought. In a cursory search of his reported cases, I find no discussion of infections as a causative factor in mental defect. The standard English textbooks of psychiatry[4] either make no mention of the matter or refer to it briefly.

Occasionally one finds some attempt at statistical solutions. Thus, in Piper's[5] statistical compilations there are reported seventy-five cases of acquired idiocy without convulsions: 29 per cent. were due to scarlet fever and diphtheria, 12 per cent. to measles and 8 per cent. to encephalitis. Of thirty-two cases of acquired idiocy with convulsions, 22 per cent. were traced to scarlet fever and typhoid and 10 per cent.

1. Tredgold, A. F.: Mental Deficiency, New York, William Wood & Co., 1908 and 1914.
2. Ireland, W. W.: The Mental Affections of Children, Idiocy, Imbecility and Insanity, London, J. A. Churchill, and Edinburgh, J. Thin, 1898.
3. Bourneville: Recherches sur l'épilepsie, l'hystérie, et l'idiotie, Paris, 1881-1901.
4. Church and Peterson, Jelliffe and White, Paton, Diefendorf, DeFursac, etc.
5. Piper, Hermann: Zur Aetiologie der Idiotie, Berlin, H. Kornfeld, 1893.

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each to measles, "gastric fever" and encephalitis. This is roughly equivalent to finding 40 per cent. of acquired idiocy due to infectious diseases. The statistics of other writers do not bear out these high figures. For example Koernig,[6] in a list of 260 cases of idiocy, carefully analyzed as to etiology, found 3.4 per cent. (he questions whether this should be lowered to 2.3 per cent.) due to infectious diseases aside from syphilis. Even in his list it ranks above traumatism as a cause, and several times greater in frequency than the oft-cited consanguinity of parents. Pearce[7] cites several tables. Beach and Shuttleworth's figures from the Royal Albert Asylum are given, showing "acute infectious fevers" to be causative in 119 of 1,200 cases. Figures for 1,180 cases from the Darenth asylum[8] are also quoted without the mention of infectious diseases! Tredgold[9] gives a table to show the postnatal extrinsic causes; of the 150 cases studied, thirty-seven cases, or nearly one-fourth, fall in this group, but of these only seven are ascribed to "infectious fever." It is surprising that so capable a writer should have ascribed no less than ten cases to "teething convulsions" [sic].

There seems to be no doubt but that syphilis may be a cause of feeblemindedness.[10] This, however, opens up too big a field for present discussion and is really beside the point, since we are here dealing with acute infections, and syphilis must certainly be regarded as chronic.

The classification of G. E. Shuttleworth and Fletcher Beach[11] of England should be mentioned.

6. Koernig, W.: Die Aetiologie der abortiven Formen einfacher Idiotie verglichen mit derjenigen der cerebralen Kinderlähmungen, Allg. Ztschr. f. Psychiat. **61**: 133, 1904.
7. Pearce, F. S.: Causes Conspiring to Produce Mental Enfeeblement in Children, Am. Med. Surg. Bull., Aug. 1, 1895, p. 908.
8. These figures are taken from Tuke's Dictionary of Psychological Medicine **2**: 659-665, 1892.
9. Tredgold, A. F.: Amentia, Mott's Arch. of Neurol., Pathological Laboratory of the London County Asylum **2**: 328-423, 1903.
10. Southard, E. E., and Solomon, H. C.: Neurosyphilis, Boston; W. M. Leonard, 1917.
11. Shuttleworth, G. E., and Beach, Fletcher: Presented at the Thirteenth International Medical Congress, Paris, 1900, and embodied in their article in Allbutt and Rolleston's System of Medicine, London, the Macmillan Company, 1910, Vol. 8.

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In general, it consists in grouping the forms of mental defect according to etiology, as congenital, developmental and accidental. Waiving the obvious deficiencies of a scheme which does not foresee the conflict between the second and third groups as given and named, I shall proceed to the minutiae of the third group, with which we are now concerned. They recognize under the "acquired" or "accidental" etiology group a traumatic form, a sclerotic form, and a postfebrile form. Again, passing the first two items of a series of three (one is struck by the trinitarian tendencies of the writers), we read that this is "another important type;" and measles, pertussis, typhoid and scarlatina are specifically mentioned.[12] These authors proceed then to the pathogenesis. This subject is discussed in greater detail by other writers, e. g., Bolton,[13] Hammarberg,[14] and above all, the elaborate monograph of Fernald, Southard and Taft.[15] Shuttleworth and Beach apparently attempt to mask their study of the infectious factor by a show of words: "These diseases cause inflammation of the brain and its membranes, or are complicated with it not sufficiently grave to be fatal, but serious enough to cause mental impairment." Added to this vague affirmation they quote Ireland as saying "Truly" the following banality: "The amount of damage to the intellectual powers must be mainly dependent upon the intensity of the morbid process." If by the phrase "morbid process" Ireland meant the systemic disease supposed to precipitate the hypophrenia, we know that in the case of influenza, at least, he was utterly incorrect, since it is certainly quite generally agreed that the severity of the mental sequelae of influenza bears no relation

12. Tredgold (Footnote 9) adds smallpox and syphilis.
13. Bolton, J. S.: The Histological Basis of Amentia and Dementia, Mott's Arch. of Neurol. 2: 424-620, 1903; On the Brain in Health and Disease, London, E. Arnold, 1914.
14. Hammarberg, Carl: Studien über Klinik und Pathologie der Idiotie, Upsala, E. Berling, 1895, p. 126; Leipzig, Koehler.
15. Fernald, W. E.; Southard, E. E., and Taft, A. E.: Waverly Researches in Pathology of the Feebleminded, Mem. Am. Acad. of Arts and Sciences 14: (May) 1918.

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whatever to the (apparent) severity of the clinical symptoms of the disease.[16]

Another statement of the writers is more worthy of note, ambiguous though it be: "Of course, if one of the diseases we have mentioned should occur in a child previously disposed to imbecility [sic] this affection is more likely to follow it." One is not at all sure just what the writers meant, but perhaps it was to signalize the process of aggravation or intensification of preexistent hypophrenia by acute infectious disease.

METHODS OF STUDY

What is the effect of influenza *re* feeblemindedness? This is the specific question of my research. More minutely, it is to learn whether or not influenza does or can produce frank mental defects, and whether or not it alters for better or worse the mental status of patients with an already existent mental defect. These questions are really put to us, not by the influenza epidemic, but by the progress of study and research concerning hypophrenia. The influenza epidemic has contributed data to their solution.

The collection of these data was thus made: In the first place, to insure a wide sweep of material, questionnaires were prepared and sent to the superintendents of all state institutions for mental defectives except those located in states where the influenza epidemic was inconsequential.

About 75 per cent. of all the letters were answered. These answers were in general kindly and carefully made, in most cases a letter of particulars being sent. There was no equivocation or evasion in the replies. The total number of institutions replying was seventeen. I appreciate the cooperation and help of the institutions that replied, which are:

Rhode Island School for the Feebleminded, Slocum, R. I., Dr. Joseph H. Ladd, superintendent.

16. This point has been made by numerous writers, e. g., Jelliffe, Menninger, Althaus and Fell.

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Massachusetts School for the Feebleminded, Waverly, Mass., Dr. Walter E. Fernald, superintendent.

New Jersey State Institution for the Care and Training of Feebleminded Girls and Women, Vineland, N. J., Dr. M. A. Hallowell, superintendent.

State of New York Letchworth Village, Thiells, N. Y., Dr. Charles S. Little, superintendent.

State Institution for the Feebleminded of Western Pennsylvania, Polk, Pa., Dr. J. M. Murdich, superintendent.

Nebraska Institution for Feebleminded, Beatrice, Neb., Dr. D. G. Griffiths, superintendent.

Institution for Feebleminded, Columbus, Ohio, Dr. E. J. Emerick, superintendent.

Syracuse State Institution for Feebleminded Children, Syracuse, N. Y., Dr. O. H. Cobb, superintendent.

The State Home and Training School for Mental Defectives, Ridge, Colo., Dr. A. P. Busey, superintendent.

Sonoma State Home, Eldridge, Calif., Dr. F. A. Butler, medical superintendent.

Wisconsin Home for Feebleminded, Chippewa Falls, Wis., Dr. A. W. Wilmarth, superintendent.

Huntington State Hospital, Huntington, W. Va., Dr. L. V. Guthrie, superintendent.

Oregon State Institution for Feebleminded, Salem, Ore., Dr. J. N. Smith, superintendent.

State Institution for Feebleminded, Medical Lake, Wash., Mr. S. C. Woodruff, superintendent.

State Custodial Asylum for Feebleminded Women, Newark, N. Y., Dr. Ethan A. Nevin, superintendent.

Rome State Custodial Asylum, Rome, N. Y., Dr. Charles Bernstein, superintendent.

State Home for Feebleminded, Winfield, Kan., Mr. Wylie W. Cook, superintendent.

The first question asked was: "How many, if any, cases of mental defect or feeblemindedness were admitted to your institution since the recent epidemic in which the influenza was given as a precipitating or contributing factor?"

To this question there was a unanimous answer of "None." If the average admission rate holds for the institutions from which replies were received, this is equivalent to saying that of more than 500 new patients with mental defects admitted since the influenza epidemic to seventeen leading state institutions for the care of these patients, in not a single instance was influenza even mentioned as a precipitating or contributing factor.

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The second question was: "How, if at all, were the patients of your institution, who had influenza in the recent epidemic, affected by that disease as to possible amelioration or augmentation of their mental defect?"

Of the seventeen replies, fifteen were unanimous to the effect that no amelioration or augmentation of symptoms was observed, or at least none that could be made sure of or was worthy of note. One writer spoke of psychotic sequelae, a matter to be taken up later in this paper. Another writer (Dr. W. E. Fernald, of the Waverly School) writes that while "as a rule" the patients were unaffected by influenza as far as mentality was concerned, "one patient, an old meningitis case, improved very much after her acute symptoms subsided." There was frequent mention of the frequency of tuberculosis in patients after influenza, and some comment on altered physical states, but no other observations of altered mental state or capacity.

To summarize: Of seventeen institutions with a total population, according to the figures of the superintendents, of over 16,000, in only two institutions and in one of them in only one case, was any relation observed between influenza and mental defect. Surely one might consider this a definite answer to the proposed questions.

SECOND METHOD OF STUDY

As a matter of fact, the surprisingly barren results of the first method are probably misleading. The fallacy may lie in the inattention to details which the pressure of executive work, influenza itself and shortage of professional help combined to make inevitable. It may lie in the crudity of our standards of comparison, our clinical psychometry. It may lie in the wording of the questions.

But wherever the location of the fallacy, fallacy we must believe it to be in the face of data acquired from

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scanning specific cases encountered in private practice and neuropsychiatric dispensaries and hospitals. From such sources the cases reported herewith are taken.[17]

These cases of hypophrenia influenced by influenza may be divided for the purposes of study into six groups, three under the heading of "Simple or Uncomplicated Hypophrenia," and three under "Complicated Hypophrenia." All but two patients of the series were hypophrenic to begin with. Thus we possess no definite evidence that influenza can produce hypophrenia (as distinct from a hypophrenic, i. e., demented, form of psychosis, which is of course beyond dispute). Nor does the literature help us on this point. The only reference whatever to the matter which I could find after a careful search is this from Paton's [18] textbook:

> Not a few children, born healthy, after a severe attack of diphtheria, *influenza,* measles, scarlet fever, typhoid fever or meningitis, are left mentally deficient.

The following cases are presented, not to illustrate alone the formula:

$$\text{Normality} + \text{Influenza} = \text{Hypophrenia}$$

(although two of the cases might be so represented), but perforce to illustrate the various forms of conclusion to the paradigm;

$$\text{Hypophrenia} + \text{Influenza} = X$$

Under the caption of Simple or Uncomplicated Hypophrenia (as opposed to hypophrenia with complicating factors or tendencies), there are three groups, easily separable on the basis of Southard's [19] ingenious scheme for labeling the qualitative variations in the

17. Of the following cases, six are from our Boston series (Menninger, K. A.: Psychoses Associated with Influenza, J. A. M. A. **72**: 235 [Jan. 25] 1919), from which previous studies have appeared (the reference just given; Footnote 16, and Menninger, K. A.: Influenza and Neurosyphilis, Arch. Int. Med. **24**: 98 [July] 1919). Three were reported in a letter to the writer from Dr. Joseph H. Ladd of the Exeter School of Rhode Island, at Slocum. Two of the patients were seen in private practice in this state (Kansas), one by Dr. O. S. Hubbard, superintendent of the Kansas State School for Epileptics, in consultation with another physician, and one by the writer in consultation with Dr. W. S. Lindsay, of Topeka, Kan. To all of these men credit is herewith duly awarded, and an expression of gratitude proffered for their kindness in putting at my disposal the data on the cases that I did not see.

18. Paton, Stewart: Psychiatry, Philadelphia, J. B. Lippincott Company, 1905, p. 241.

19. Southard, E. E.: Suggestions in the Nomenclature of the Feeblemindednesses, Mental Hygiene **2**: 605 (Oct.) 1918.

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psychic constitutions of hypophrenics. The groups are: (1) those with intellectual sphere disturbances conspicuous; (2) those with emotional disturbances uppermost, and (3) those with conduct disturbances prominently intensified after influenza. Or, to use Southard's nomenclature, these three groups might be thus labeled:

Group 1. Hypognosis = the prominent psychic product of influenza.
Group 2. Dysthymia (parathymia or hypothymia) = the prominent psychic product of influenza.
Group 3. Parabulia = the prominent psychic product of influenza.

The complicated cases require a somewhat different approach. They, too, fall into three classes:

Group 4. Hypophrenia with neurologic manifestations prominent.
Group 5. Hypophrenia with psychic manifestations prominent.
Group 6. Hypophrenia with psychopathic manifestations prominent.

These, like the elements of the preceding group, may be abbreviated, as:

Group 4. Hypophrenia with encephalopathy.
Group 5. Hypophrenia with psychosis.
Group 6. Hypophrenia with psychopathy.

Each of these six groups will be taken up separately and illustrated with the appropriate cases.

SIMPLE HYPOPHRENIA: GROUP 1. WITH INTELLECTUAL DISTURBANCES CONSPICUOUS

Hypophrenia with disturbances (loss) in the intellectual sphere prominent following influenza (or hypophrenia, with hypognostic aggravation by influenza) is, of course, the only type in which the demonstration of mental loss by such mathematical means as the Binet scale and its descendants is possible. The thrill afforded the psychologist in finding the mental age of a child to have fallen so many points after influenza can never be equaled in a cold, critical psychiatric study of conduct or affect before and after.

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Case 1 fits well into this group, although, of course, as usual, there was an associated disturbance of conduct and emotional balance. This case is reprinted with abridgment from my expository analysis of the postinfluenzal mental cases, where it was given to illustrate the hypophrenic group.[20]

CASE 1.—*Abstract.—A lad, aged 10, of American parentage, previously very dull in school, but well enough behaved, after an attack of influenza became much disturbed and was brought to the hospital for care. When his excitement subsided it was found that his intellectual level had apparently fallen even lower than before. The diagnosis, on admission, was low grade moron; on discharge, high grade imbecile. The effect of influenza was aggravation of the hypophrenia, all spheres, but particularly the intellectual.*

History.—The family history was entirely negative. An only brother, a year younger, was quite well and was doing fair work in the fifth grade. The patient was born at full term, normal delivery, and had had no severe illnesses. His teeth appeared at 5 months; he walked at 18 months, talked at 3 years, and started kindergarten at 4 years. He did poor work in kindergarten the first year and was kept there a second year, which was done only "with particularly backward children." He was promoted to the first grade but had to repeat the work, and then, though it was still poor, he was promoted. "His work in the second grade was poor, but he was promoted because considered too old to stay in the grade." The third grade work was poor, and was repeated this year . . . record, so far, poor. The patient had so far had the very lowest rank given, and was promoted because of a desire not to keep backward children behind indefinitely." He had always been quiet and rather seclusive, and played with childish toys which even the younger brother had outgrown. His teacher reported that his deportment was good, and that he did not seem sensitive about his backwardness. There had never been any conduct disorder of note.

Present Illness.—He had had influenza for four days; the temperature was not known. He had "slept without waking" for three days, taking no food. The physician and the nurse insisted on his staying in bed a week, which he did without complaint. He was "perfectly normal and happy" up to this time. Three weeks after the influenza (on Christmas morning) he complained of headache and fatigue, and according to his parents' statement "had been out of his head since." (The history was obtained on the 28th.) He cried

20. This point has been made by numerous writers, e. g., Jelliffe, Menninger, Althaus and Fell.

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constantly; wandered about aimlessly; went outdoors and became lost; occasionally sang in a feeble voice numerous popular songs; at other times screamed and cried and refused to remain in bed; said, "I'm lost; take me home; I will be good to you." His parents were alarmed because he failed to recognize his mother. He was brought to the outpatient department and recommended to the house.

Mental Examination.—For the first two days, he blubbered and wailed constantly, the more so when any attempt was made to comfort or quiet him. He selected one of the nurses finally, whose attentions he would permit, and dragged himself about the ward at her heels thereafter, crying when anyone else approached him. He was sleepless for the greater part of the first four nights, but gradually slept better at night and cried less in the daytime, until on the fifth day he was quiet all day, clothed, accessible and in a faint way cooperative. Orientation for place, time and person was retained from the start. His memory defect was most prominently shown in school learning. He was tested by the tests used at the Waverly School for the Feebleminded and found incapable of doing any of the third grade tests, many of the second grade tests, and not a few of the first grade requirements.[20] There were no amnesic periods, no delusions or hallucinations, and no defect of attention after the fourth day. The associations were rather slow but not schizophrenic, incoherent or erratic, although, of course, quite puerile. Emotional tone was, of course, in question. During the examination he made no display of emotion except at the mention of home, which evoked a few quiet tears, and on the day of discharge he was quite elated and happy because he was going home. His conduct disorder was confined to the wailing and screaming of the first few days.

Subsequent History.—On the day of discharge, January 4, his lugubriousness was replaced by smiles and complacency. His memory was again tested briefly and found impaired, even for the recent episodes; insight was essentially lacking, although he did say that his head was "not right" and that he had been dizzy from rocking too much; the intellectual processes were definitely but slightly improved; ideation was as before, and conduct was above reproach. He took no interest in trying to do ward work and gave no other manifestations of normal initiative.

Physical Examination.—He was a rather undersized lad, whose physical examination was negative in all respects.

Laboratory Findings.—The urine was negative. The blood serum Wassermann test and spinal fluid tests, including the Wassermann and Lange tests, were all negative.

20. Some of the most glaring deficiencies were: He could not count by two's, he could not do subtraction at all, he misspelled "do," "go," "run" and other words little more difficult. He did spell correctly "cat," "it," "and" and "an," and did a few simple problems in addition, such as "2+3, 1+4, and even 3+7," but could not get much above the latter.

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Psychometric Tests.—The patient graded regularly, with a variation total of 7, at a mental age of 5.7 on the Yerkes Bridges point scale. On the Stanford scale, four days later, he graded at 6 years, 2 months. In the supplementary tests his performance in the construction puzzles was poor. The memory tests were also poorly done, and he accepted ten out of ten suggestions. The patient cooperated poorly on the point scale and fairly well on the Stanford.

Final Diagnosis.—He was discharged with recommendation for instruction in a school for the feebleminded. The diagnosis at discharge was imbecility.

GROUP 2. WITH EMOTIONAL DISTURBANCES

Cases 2 and 3 are illustrative of predominant dysthymic manifestations of hypophrenia as aggravated by influenza.

CASE 2.—*Abstract.—A negro lad, aged 16, who, although always regarded as peculiar, had never been troublesome or disagreeable, after an attack of influenza became so inexplicably irritable, sullen and irresponsible that his father took him to court as a "stubborn child that hasn't good sense," to "have his mentality taken." The diagnosis was hypophrenia, low grade moron. The effect of influenza was distinct aggravation, particularly of emotional disturbances.*

History.—The mother was an epileptic, who had had influenza in September and presumably in her delirium, had jumped from the roof of her house, killing herself. The father seemed to be above the average negro, and had had a university education. There was no history of any nervous or mental trouble among the six siblings. The patient was born in Boston and had attended kindergarten at the age of 5. He left the fourth grade at the age of 16, after having been held back in every grade. At the age of 5 or 6, he was "vaccinated" for diphtheria during an epidemic, but contracted the disease nevertheless, and "had never been the same since." He was always regarded as peculiar, even by his parents. He seemed to have been a sociable lad and was fond of going to parties and dances. He wandered about a good deal and could not succeed in keeping any job more than a few weeks. Apparently he worked well, but objected to having his father take his wages ($9). However, until the present illness he had never been in trouble of any kind, nor was there any record that he was ever suspected of being feebleminded or insane.

Present Illness.—In September, the patient had influenza. He was ill for two weeks and was delirious for two or three days. Both the boy's father and the boy himself dated the onset of his present trouble from that event. In the course of the examination, the lad said, "Since I had the flu I ain't

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been well, and people tell me I am wrong in the head. I don't get along as well as I used to, especially at home." The father's account was that since he had had influenza he had become very stubborn, wayward, distractible and irresponsible (these are almost the father's exact words). His emotional balance had become very much upset so that while occasionally cheerful and agreeable he was most of the time sullen, morose and irritable. He took to sitting alone in the dark for hours, idle and abstracted. The father finally, in desperation, took him to the police station and swore out a warrant against him as a stubborn child. He affirmed in court that the boy "hadn't good sense." It was from this court that the boy was sent to the hospital for examination.

Examinations.—Mental: The mental examination confirmed very well the father's report of the child's emotional disturbances and his volitional and intellectual deficiency. He was at first not at all cooperative or attentive, and was sullen, restless and ill at ease. Later he became more cooperative and accessible, and talked pleasantly in the ward. He became quite panic stricken on contemplation of the rumored lumbar puncture. Later he was somewhat obstreperous about being confined in the hospital. His attention was never very good, and his thought processes showed a general scattering and weak associations. His thought content showed a paucity and inadequacy in the ideational realm, but there were no delusions, obsessions or phobias. There were no hallucinations in any sphere. His orientation was only approximate; his memory for remote events was quite poor and for recent events quite inaccurate. He had very little or no insight, although he said, "They claim I am wrong in my head."

Psychologic: "The patient graded irregularly with a variation of 17 at a mental age of 8.7. In the supplementary tests, he did very well in the memory test. Nine out of ten suggestions were accepted. He cooperated well."

Physical: Thorough physical and neurologic examinations were entirely negative. The blood pressure was: systolic, 118; diastolic, 70. He was 5 feet, 2½ inches tall, and weighed 120 pounds.

Laboratory: Examinations of urine, blood and spinal fluid were entirely negative.

CASE 3.—*Abstract.—A teamster, aged 29, apparently a typical moron, without previous episodes of emotional or conduct disturbance, after influenza, diagnosticated in a base hospital, became disinterested in life and melancholy to the point of attempted suicide. The diagnosis was hypophrenia (moron grade) with depression. The effect of influenza was aggravation, particularly of the dysthymic symptoms.*

History.—The family history, which was obtained in detail, was entirely negative, including an account of the parents and of the paternal and maternal siblings. The parents were as

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ignorant as the average Irish immigrants, but the six siblings were all progressive and self-supporting. The father was described as being sociable, good natured, but quick tempered, and nonalcoholic; the mother as having a quiet, friendly, even disposition. Both parents and siblings were well, and the only death in the family was that of a year old brother. The patient was born in a Boston suburb, where he started to the parochial school at the age of 5, and after 10 years achieved only the fourth or possibly the fifth grade. The reason for his leaving may have been his failure to progress, although the brother, who seemed to be a man of some intelligence, ascribed it to extraneous conditions, poor teachers, insufficient books, etc. No conduct disorder was alleged. After leaving school, he served as errand boy for a grocery (at $9 a week) for a few years, became a teamster, and had remained at that work more or less continuously since. It was hard for him to hold a job very long. For a year and a half before being drafted, he worked for an ice company at $3.25 and two meals a day. He started drinking when about 17, and had kept it up steadily since, periodically and distinctly to excess. He had been arrested repeatedly for drunkenness. He smoked more than twenty cigarets a day. He had never been married, and denied excessive sexual irregularities and venereal disease. He was regarded by his family as "spendthrifty." The brother and father said: "He has a quiet, good-natured disposition, and has many friends. He is naturally lazy and inclined to be selfish. He likes to go to movies and theaters, and read newspapers. He has never been considered as nervous in disposition, but is rather staid and serious."

Present Illness.—The patient, for some unknown reason, steadfastly denied having had influenza or any other illness. Nevertheless, the facts seem to be that about six or seven weeks before admission to our hospital, he was in a U. S. Army base hospital with the ubiquitous influenza, so diagnosed, where he remained for twelve days. Shortly after his recovery he was discharged, physically well, because of the demobilization. He had seemed to his relatives, with whom he lived, to be unusually quiet, ambitionless and depressed. "He kept saying he would like to be in the good condition he was in before going into the army. He was sad, could not work, . . . tried it one day, but felt too weak." Apparently his depression increased, and on the day before admission he attempted suicide by turning on the gas. He stated afterward that this was a subterfuge of his own device to gain admission to the hospital (!). He was taken to the police station, revived, and advised to go to the psychopathic hospital by the physician called. This he willingly did.

The mental examination confirmed the diagnosis suggested by the history. The psychometric age was 9.3. He recognized his own mental inferiority. There were, in addition to

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the usual shallowness of thought processes, the paucity of ideas, puerility of judgment, etc., of hypophrenia, and symptoms of depression with retardation of thought and movement.

The physical examination and laboratory findings were negative.

The emotional disturbances which the influenza seems to have provoked in these cases are perhaps analogous to the psychic discomfiture, the depression, irritability, languor, and sometimes mild agitation or melancholy which is so often seen in (supposedly) normal persons after an attack of influenza. I have previously pointed out [21] that, whereas these depressive symptoms, or a depressive tendency, are very frequent in normal persons (some say they are always present, e. g., Pritchard,[22]) they are comparatively infrequent in persons who develop psychoses. The observation has been disputed by Fell [23] but without sufficient substantiation by clinical data.[24]

GROUP 3. WITH CONDUCT DISTURBANCES

Conduct and volitional disorders are so constantly associated with intellectual lack in hypophrenia that one might expect a postinfluenzal volitional disturbance in some hypophrenics. Such is actually the fact, and these cases are apparently the most frequent of all. Perhaps it is that these known hypophrenics

21. Menninger, K. A.: Psychoses Associated with Influenza, I, A Statistical Analysis, J. A. M. A. **72**: 235 (Jan. 25) 1919.

22. Pritchard, W. B.: Nervous Diseases and Psychoses Following the Grippe, Internat. Clinics Series V, **11**: 1.

23. Fell, E. W.: Postinfluenzal Psychoses, J. A. M. A. **72**: 1658-1661 (June 7) 1919.

24. In the hypophrenic patients there has not been noticed any general tendency to depression following influenza. The usual cheerfulness is in most cases apparently retained. Nor have I heard from the superintendents of the institutions caring for the feebleminded that irritability, moroseness or truculence were increased after the epidemic. On the other hand the emotional disturbances in the hypophrenic patients whose mental state was distinctly altered by influenza in other ways is noteworthy. The striking manifestation of it in Case 3 is able evidence. Case 4 is even more pertinent, since the dysthymia is the outstanding feature. Consequently, one is led to deduce that the hypophrenic patients follow an inverse law to that applied to normal and psychotic individuals. These two principles may be thus contrasted: Most normal persons suffer emotional depression after influenza, but of those in whom the influenza precipitates a psychosis, few exhibit depression. Most hypophrenic patients do not suffer depression after influenza, but of those in whom influenza produces marked mental changes, the majority do exhibit emotional disturbance (depression or irritability, etc.).

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whose psychomotor control becomes impaired to a noticeably greater extent are only more conspicuous than the cases of intellectual deterioration, or those emotionally disturbed. The fact of the greater ease of recognition may account in some degree for their greater frequency. At any rate, we have been able to collect the reports of several cases from the recent epidemic, one of which (from our Boston series) appears below. Three of the cases mentioned by Burr [25] would probably fall in line here.

CASE 4.—*Abstract.—A white girl, aged 17, clearly subnormal, of a hypophrenic family, whose conduct disorder became so remarkable after an attack of influenza that she was brought to the psychopathic hospital for examination, was diagnosed as being hypophrenic (of subnormal grade). The effect of influenza was distinct aggravation of symptoms, particularly conduct and emotional disorders.*

History.—The patient, who was born in Boston of New England parentage, had two brothers in state institutions for the feebleminded, and five other siblings were in the public schools. Her physical life had always been excellent, and there were no stigmas of nervous or epileptic disease, such as fainting attacks or enuresis. She attended school from 6 to 13, and progressed to the fifth grade. At that age she was committed to the state reformatory for girls because of her extravagant sexual promiscuity. There she remained through a stormy period of four years. She was given a place to work after her discharge from there, but lost it through a sexual exploit. A second place was secured which she voluntarily gave up because of disinclination to do the work, saying that she was engaged and did not need to work much longer.

Present Illness.—The girl had influenza late in the fall. Subsequently, she became "very ugly and irritable," charging unjust treatment, and was evidently highly exasperating (and so impressed the examining and admitting physicians at our hospital). She had talked much of her wish and hope to die, and "exposed herself again to influenza" in the hope of attaining that result. She capped the climax by sending for the physician and then running away to a cinema before he arrived. The parole board sent her to the psychopathic hospital for examination.

Mental Examination.—The prominent features of the psychic examination were the emotional instability and the

25. Burr, C. W.: Mental Complications and Sequelae of Influenza, Med. Clin. North America 2: 709 (Nov.) 1918.

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intellectual lack. She was extremely antagonistic at first, irritable, impertinent and quite inaccessible. She talked constantly, but refused to answer questions. Later she made herself quite agreeable and cooperative. Her ideation and judgments showed the typical childishness and inadequacy. Many of her statements were of questionable reliability and veracity. There was a slight paranoid coloring to her story, as is frequent with the delinquent hypophrenic sent by the parole board for examination. The psychometric test gave her a mental age of 12 years, with a variation total of 11. "The patient cooperated well (!). Her performance on the construction puzzles and on the memory tests was good. In the suggestibility test she resisted all ten of the suggestions offered!" (The latter is interesting in view of her reputed immorality, etc.). Her retention of school knowledge was very poor, indeed, except in arithmetic, in which she did only fairly well.

Physical, gynecologic and neurologic examination, and analyses of urine, blood and spinal fluid proved negative.†

COMPLICATED HYPOPHRENIA

The preceding cases of the three groups have all been examples of simple, idiopathic mental defect without psychiatric or neurologic complications. Now follow cases of hypophrenia, of mental defect to be sure, but cases in which there are neurologic or psychiatric elements that are conspicuous enough to determine their place in the list.

The importance of this factor of conspicuousness, to digress, is often overlooked for the very inconspicuousness of itself. Consider an imbecile child in an institution for the feebleminded. What though an ambitious and hypercritical clinician establish the fact that this child's spinal fluid gives weakly positive Wassermann and gold reactions? The child continues to be regarded and treated as an imbecile, that is, as hypophrenic, although it may be technically conceded on the records that he is a neurosyphilitic imbecile (which is, of course, probably incorrect, as he would likely be an imbecilic neurosyphilitic). But, on the other hand, if the child, however hypophrenic,

† The case of improvement of uncomplicated hypophrenia given below would logically fall in this group of simple hypophrenia, but it deserves the emphasis of later consideration.

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developed peripheral gummas, or if the father became notorious as a paretic, etc., there is a likelihood that enough conspicuousness might be thrown up about the little imbecile to secure for him the proper label, and thus the proper classification, and thus the proper treatment. Conspicuousness, then, may determine classification, and hence treatment.

It is astonishing to hear certain otherwise intelligent persons rail against the classifications and the attention paid to classifications, particularly in psychiatry, when for want of them patients continue to be misplaced and mistreated or not treated at all, throughout the whole country. It is safe to presume that there is no state in the Union whose penal institutions are free enough from hypophrenics, or whose feebleminded institutions are free enough from neurosyphilitics, patients with brain tumor, etc., to throw stones at this deposition. Herein lies the incalculable advantage of correct and applied classifications, and the service of Kraepelin, and more recently of Kirby,[26] of Southard[27] and of Orton.[28] The work of Fernald, Southard and Taft in carrying on the projects of Bourneville and Hammarberg to ascertain the actual pathologic why of hypophrenia is likely to achieve great results through the machinery of a classification, and if we are to look forward to a *mundus sine hypophrenia,* surely this is our most promising route.

For these reasons, then, the following cases cannot justly be left in a group of "simple hypophrenia"; and in the present imperfect state of our knowledge of that affection, to say nothing of our ignorance of the complications of hypophrenia, a classification must be framed with the greatest possible generality of terms. In every instance, of course, there is a hypophrenic

26. Kirby, G. H.: Manual Prepared by the Committee of the American Medico-Psychological Association.
27. Southard, E. E.: The Genera in Certain Great Groups or Orders of Mental Disease, Arch. Neurol. & Psychiat. 1: 95 (Jan.) 1919.
28. Orton, Samuel: Classification presented at the 1919 session of the American Medico-Psychological Association, and appearing in the transactions of that meeting.

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factor, and its precise relationship to the complications varies in each case.

The first two cases constitute a group I have labeled "Hypophrenia with Neurologic Manifestations Prominent." Neurologic symptoms being conspicuous, these patients would be suspected of being encephalopathic before being suspected of being hypophrenic. As will be seen from the data presented, however, both cases make bids for both the neurologic and hypophrenic groups, for the yet tenuous group of hypophrenia on organic bases, of this sort and that sort. These two cases may be examples of feeblemindedness directly resultant from influenza.

The next four patients were regarded as unquestionably hypophrenic before they had influenza. After influenza, psychoses developed. Nor were these psychoses the familiar episodic interruptions of the otherwise placid course of the average hypophrenic's existence. They were, apparently, frank and severe psychoses. Our knowledge of the psychotic manifestations of which hypophrenics are capable is so barren that it is futile to attempt a psychiatric diagnosis of these cases.

The third group comprises two cases, not obviously but probably hypophrenic, and certainly psychopathic. I should like to avoid the use of the ambiguous and cumbersome phrase "constitutional psychopathic inferiority" and others applied to the same state, but the identity of the picture we have in mind is indisputable. The influence of influenza on these cases is equivocal. I believe that it was definite, however slight. I admit, however, that there is room for an opposing view from the self-same data. They are offered at their face value.

GROUP 4. WITH NEUROLOGIC SYMPTOMS CONSPICUOUS

CASE 5.—*Abstract.—A girl, aged 4 years and 4 months, with a father and two half-brothers with notably large heads, who, although not very bright, had never been considered particularly dull, and certainly never in ill health, prior to an exceedingly light attack of influenza, subsequently developed*

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an intention tremor and a flaccid triplegia, incontinence, hypotonia, absent abdominal reflexes, positive Babinski reflex, neuroretinitis and a marked mental enfeeblement. A diagnosis of gross brain disease (Multiple sclerosis? Cerebral neoplasm or abscess?) was made. The influenza was apparently causative.

History.—Two aunts and two cousins had died of tuberculosis; a paternal uncle had suffered from periodic headaches. The father wore a 7⅝ hat, and had a massive head and body. Two other children of the same mother, but by a different father, were remarkable for their large heads. One boy, aged 9, wore the same sized hat as his foster-father, and his 14 year old sister also had a large head (these were half-siblings of the patient). There was no history of nervous or mental disease in the family. The child was born normally, at full term, and was breast fed for one year. There were no convulsions in infancy, and teething, talking, walking, etc., were not delayed. She had always been a healthy and apparently normal child. There was nothing to prove that she had been hypophrenic before the influenza. She had learned a few rhymes and was able to repeat them, even during the present illness. Her parents thought her "as bright as the other children," and no doubt was entertained as to her or their mentality.

Present Illness.—The child had influenza in December, 1918, and remained in bed a week, although she was "not sick at all and it was like punishment to keep her in bed." About March, the parents began to notice a "very slight trembling of the hands when she went to use them." When they were quiet, this was absent. This tremor increased very slowly. But in April she began to be "a little wavy" and unsteady in her walk, with "not quite so firm a step as usual." Both these symptoms increased in severity. Vomiting attacks, never projectile, but of all degrees of severity, came as the next symptom. They recurred for a time about once in ten days, but with no regularity. At this time, also, she had mild headaches, but these were never severe, nor did the child ever complain of any pain, or show symptoms of a psychosis.

May and June were uneventful, save for a steady though slight increase in the symptoms described. She began to be unable to walk on rough ground, then unable to walk even in the house save by clinging to chairs, etc., and finally not at all. But the arms, meanwhile, got no worse after June, "and may be a little better." (With Dr. Lindsay, I saw the child in September.) The intention tremor remained very noticeable, so that she spilled water and food in her lap when she ate, etc.

During July and August there was little further change, except incontinence of urine and feces. She became entirely bedridden, but gained weight, vomited much less frequently, and rarely complained of headache and then only casually.

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Examination.—Several factors interfered with the examination, but the chief findings are recorded: The child lay apathetically on the examining table, smiled occasionally, cried when hurt, spoke very little or none at all, answered no questions, and was distinctly apathetic, disinterested, torpid and dull. A psychometric test was, of course, impracticable, but she would surely have rated no higher than 2 years.

Physically she was without defect, being excellently developed and nourished, the heart and lungs being normal, etc. The only abnormalities were those noted in the neurologic examination.

The neurologic examination revealed a very large head (the parents insisted no larger than always, and no larger than her half-brother's), with Macewen's percussion sign very marked (according to Leftwich,[29] Oppenheim and others an indication of massive intracranial disease); pupils reacting promptly but insufficiently to light; absent abdominal reflexes; bilateral Babinski reflex; occasionally elicited Oppenheim and Shaffer signs; a very marked hypotonia, and an angry retina with normal sized vessels but with curious disks which appeared to be in a state of postneuritic atrophy. In addition, there was an intention tremor of the left arm which was unmistakable; the right arm could be used with good direction but with little force, and both legs could be guided, but could not sustain weight.

Examination of the blood and spinal fluid, and of the urine, all proved wholly negative. Six Wassermann tests on the spinal fluid were negative. The cell count was 1; the globulin test was not reported.

A roentgen-ray examination disclosed a large but vague shadow located centrally in the brain, and marked evidences of pressure. (The spinal fluid was not under pressure at time of withdrawal.) Whether or not the roentgen ray confirmed the diagnosis of tumor or of internal hydrocephalus was equivocal.

The diagnoses of cerebral neoplasm or abscess, and multiple sclerosis are both supported by some of the findings and contradicted by others. The fact of the presence of marked mental impairment was obvious, and for that reason the case was included here.

CASE 6.—*History* (from a letter by Dr. O. S. Hubbard).—"Baby C. was seen in consultation. Eleven months old. Heredity, siblings and past history negative. History of enlargement of head since attack of influenza. Rather poorly nourished, with a leaking heart, and a well marked hydrocephalus. The child had the characteristic enlargement of the head with the large area between and above the eyes. The child seemed considerably reduced mentally, and was afraid of strangers, could not use its body much, and appeared to be in pain when moved."

29. Leftwich: Index of Symptoms, London, 1915.

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"The doctor who referred the case did not attend the child during the influenza, but the family was suffering from the disease when the child became sick and there is no good reason for doubting the nature of the original sickness. The child has not been well from that time, and my last report is that the head is probably getting larger."

GROUP 5. WITH PSYCHOSES

Psychotic episodes in the course of feebleminded-ness are so familiar and so frequently observed that it is rather remarkable that this should be a matter so little investigated or understood. It is not even a matter of knowledge, for example, whether or not an imbecile is capable (with or without a psychosis) of hallucinating. (I am indebted to Dr. E. E. Southard for this suggestion.) It is far from established what general form the psychoses with hypophrenia take, or what their nature or tendency. In fact, one thing alone seems to be established, namely, that psychotic manifestations in hypophrenics are usually episodic and transitory.

The precipitating causes of even these episodic displays are little enough understood. It seems likely that both physical and psychic factors may act. Thus "Zip," a notorious case described (with necropsy) by Fernald, Southard and Taft,[14] died during what appears to have been a psychotic episode precipitated by psychic pain (chagrin, anger, etc.). As for physical (or should we, after R. C. Cabot,[30] utilize the word exophysical?) causation, the following cases are apropos.

Case 7 was to have been an elaboration of the case reported by Burr.[25] A communication from Dr. Burr, just before mailing this manuscript, informs me that further data are not available. I quote from his article, just cited: "A girl 14 years old, who had been simply feebleminded, became violently delirious during the fever, hallucinatory and delusional later, and finally had to be sent to an institution, where she now, three

30. Cabot, R. C.: Discussion of Southard's classification before the Congress of American Physicians and Surgeons, Atlantic City, 1919.

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months later, remains. She is dirty, destructive, foul-mouthed—a complete imbecile." (It is not altogether clear what Dr. Burr means by the adjective "complete" in the last sentence.)

Harris and Corcoran,[31] in a recent article dealing with psychoses following influenza in fifty cases, report one instance of a psychosis in a hypophrenic. The case as summarized in the original article is quoted verbatim (Case 8):

The patient was a moron, of approximately 8 years' mental development, although the chronological age was 43. In this case no unfavorable heredity was established, but there was defective make-up. There were no previous attacks. The onset of the psychosis occurred in the height of the physical illness. The psychosis terminated in recovery. This case was mentally defective from childhood and incapable of progress. She had convulsions in early childhood, but they ceased at the age of 17. She always remained at home with her parents and was incapable of retaining or making use of what little knowledge she could acquire. She assisted in an inefficient way at housework in her home. She contracted influenza, which was complicated by pneumonia, and was sent to a general hospital, where she remained sixteen days. While there she became noisy, obscene, profane, irritable and assaultive. Commitment followed. Her irritability was directed toward her mother, of whom she was previously fond. She threatened to kill her mother with a knife; attacked her sister's children; walked about the house calling members of the family vile names and constantly found fault with home conditions. Hallucinations were not demonstrated [Sic, vide supra]. She appeared oriented at all times, and there was no amnesia or clouding of consciousness. She recovered from this episode and returned to her home. It might be well to note that the mental symptoms developed during the height of the patient's physical illness, yet they were not characteristic of a somatic psychosis.

Waterman and Folsom [32] mention, without any comment or detail, that "of the twenty-three male influenza cases, one [was diagnosed] as psychosis with mental deficiency [and one as] psychosis with constitutional psychopathic inferiority." [33]

31. Harris, I. G., and Corcoran, David: Psychoses Following Influenza, New York State Hosp. Quart. 4: 469-477 (Aug.) 1919.
32. Waterman, Chester, and Folsom, R. P.: Psychoses Associated with Influenza, New York State Hosp. Quart. 4: 452-468 (Aug.) 1919.
33. A letter requesting details on these cases was unanswered.

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GROUP 6. WITH PSYCHOPATHY

Whatever the final decision as to the limits and constituents of the notorious "constitutional psychopathic state" et al., no one will gainsay that there is at least a very plausible resemblance, if not relation, between certain features of psychopathy and of hypophrenia. For this reason I make no further apology for introducing the following cases at this point.[34]

CASE 9.—*Abstract.—A girl, aged 13, who, without manifesting pronounced intellectual lack, had always been troublesome, untrustworthy and dishonest in the home, after an attack of influenza had a number of "fainting attacks" of dubious nature, and her conduct became more incorrigible than ever. The diagnosis was psychopathy in a (probable) hypophrenic. The effect of the influenza was possibly aggravation; the data were insufficient.*

History.—This child had been observed in the outpatient department of the Boston Psychopathic Hospital. Very little data of family and past history were obtained. She was known to have stolen money from her parents since the age of 6, and to have been increasingly incorrigible.

The mother's story was that the child had had influenza in the fall, and since that time had suffered dizzy spells. In some of these she fell, and in some of them she was unconscious for at least fifteen minutes. She was "unruly and uncontrollable at home, steals and lies."

Examination.—A mental examination at the hospital revealed the fact that she was "quite incapable of discriminating between right and wrong." Yet she admitted that stealing was wrong, but could not say why she had stolen except that she did it "on the impulse" (the examiner's phrase). She thought she controlled her temper quite well. She professed to like Dickens, and expressed a fondness for "detective stories and newspapers." The examiner (who was more freudian than psychiatrist) satisfied himself that no "sexual complex is present," but unfortunately neglected to secure a psychometric test.

Physical, neurologic and laboratory examinations were negative.

CASE 10.—*Abstract.—A French-Canadian girl, aged 15, with a long history of profligacy and immorality, but with a courteous and cooperative manner, since an attack of influenza had complained of feeling weak. Her maladjustments to her human environment may also have been somewhat greater*

34. I have already (Footnote 16) expressed doubts as to the possibility of demonstrating typical and unmixed "psychopathia post influenzam," but the following cases are unavowedly cases of hypophrenia complicated by or resembling or related to psychopathic states. The effect of the influenza is, of course, the point presented.

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since that time. The diagnosis was hypophrenia, with psychopathic elements prominent. The effect of the influenza was possibly slight aggravation, but data were insufficient.

History.—This girl presented quite a different picture, at first, from the usual hypophrenic delinquent. She was quiet, courteous, accessible and cooperative, and made a good impression. The immediate charge for bringing her to the psychopathic hospital was alleged neglect of her baby, a charge which she explained away deftly, but not adequately, without decrying or vilifying her parole officer or employer, neither of whom, however, she seems to have cared for. On the other hand, the girl came to the hospital with a long, black history of "breaking all the conventions of society since her ninth year," including the maltreatment of an illegitimate child, threats of infanticide, unsatisfactory service, "and is probably hypophrenic" [sic].

Her family history was what one might expect. The father was a garbage collector and the mother was separated from him. Her past history, in point of physical development and health, was quite negative. Of schooling she had little; she repeated the second grade, left at the fourth, and had little or none in the reformatory where she was sent for sexual transgressions, because of her confinement.

In October, while employed at housework, she was ill with influenza for two weeks, her temperature reaching 102. Since that time she had complained of being weak, and seemed to have increasing difficulty of adjustment with employers and parole officer. There were, however, no definite recorded data on this point.

Examination.—As to hypophrenia, however, there was no doubt whatever. Her retention of school knowledge was almost nil. She declared that "the United States entered the war, Nov. 11, 1918," and gave as the allies of Germany, "Australia is one, and the Dutch was in it." The Mississippi and the Charles were given as representative oceans, the change from a $2 bill after a 32 cent purchase as 69 cents. She did, however, give correctly the dates of the Civil War. Her psychometric rating was 9.8 years, with a variation total of only 8. "She did the puzzles fairly well and showed good learning ability. She did the memory tests fairly well. She accepted all of the ten suggestions in the suggestibility tests. She cooperated well."

Mental examination revealed her chief lack to be in the intellectual sphere. Physical, neurologic, gynecologic and laboratory examinations (including spinal fluid) proved entirely negative.

IMPROVEMENT OF HYPOPHRENIA AFTER INFLUENZA

To these cases of hypophrenia aggravated by influenza, I append now a most remarkable case of amelio-

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ration by influenza. This was the symptomatic improvement of a severe grade of feeblemindedness immediately following an attack of influenza. I saw this child at the Massachusetts School for the Feeble-minded at Waverley, and I am indebted to Dr. W. E. Fernald, whose name has made the name of Waverley mean so much in American neuropsychiatry, for his kindness in furnishing me with an abstract of the history of this case.

Speaking for the moment of the generic process of amelioration *ex nocentibus* as exemplified by influenza and neuropsychiatric cases, I may mention the recently reported cases of recovery of (presumably) schizophrenia by Henry Damaye.[35] Improvement in the manifestations of epilepsy are on record, and I am elsewhere presenting some cases in detail in the course of a general article concerning influenza and epilepsy. But as for simple hypophrenia, the case here presented is, I believe, the first recorded instance.

CASE 11.—*Abstract.*—Dorothy, aged 6, the daughter of college bred parents, whose maternal grandfather and his four brothers all had had "nervous breakdowns," who had a brother and a sister, the former very "nervous," the latter normal, was born with difficulty and had had a stormy infancy with pneumonia at 10 months, delayed dentition, etc. She had never learned to talk or to control her excreta. She had learned to walk after 3 years.

she was practically normal. "She sits and gazes at the end

She was admitted to Waverley when 4 years old. Physically, of a colored stick, waving it back and fourth. She gazes fixedly at the ceiling. Screams violently if crossed in her play." The psychometric test rating was 10 months (Stanford-Binet).

In October, 1918, at the age of 5, she was taken ill with influenza, followed by bronchopneumonia, and this by empyema. During the long convalescence, she began to show marked signs of mental awakening; she began to talk and to be tidy, learned to feed herself, learned the letters of the alphabet, attended kindergarten and cooperated with teacher and physicians. The Binet test rating in April, seven months after the influenza, etc., was 2 years and 4 months. In January, 1920, it was 3 years and 10 months, an improvement of 450 per cent. thus measured! And she continues to gain.

35. Damaye, Henry: Progrès méd. 34: 501 (Dec. 13) 1919.

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DETAILS OF CASE

Synopsis of Findings at Admission.—The patient was the third of three children; the first a boy, who had had periodic vomiting from 2 to 7 years and who was very nervous and irritable, but was improving; the second, a bright, active and normal girl. The father was a clergyman, and the mother was a college graduate (the mother's father was extremely nervous, he and his four brothers all had had nervous prostration and depression), was born in September, 1913. She was breast fed until 6 months old. The birth was difficult, the child being taken manually; there was malposition of the placenta, rapid dilation of the cervix with version and forcible manual pressure on the after-coming head. The patient had had pneumonia at 10 months, and at 2 years she suffered from vomiting and diarrhea without apparent cause. At 3 or 4 months she rolled her head. Her teeth began coming at 15 months (the other siblings had had teeth late, at 16 and 13 months). During an illness at 1½ years, her spine had been rigid. The illness at 10 months had also been called tuberculous meningitis. She began to walk a little after 3 years; she did not talk and was untidy both day and night.

Physical Examination.—The child was rather attractive looking, with a vacant stare, and she cried loudly whenever touched. The sight and hearing were apparently normal; the skin was smooth and clear. The feet were cold; the muscular development poor. She walked well. On the back of the left of the spine, there was a scar which looked like an old abscess scar.

Feb. 17, 1916, at the Waverley outpatient clinic, her mentality was rated at from 6 to 8 months. She reached and grasped things. She liked colored balls. She knocked blocks together and seemed to like noise. She did not walk, but pulled herself around rapidly. She had some minutes when she seemed to attend intelligently. She waved her hands or objects in front of her eyes.

Aug. 31, 1917: On the admission examination by the Goddard-Binet test she scored 1.[1] She would not obey a simple command and would not take either candy or wood but screamed if they were brought near to her. She tested 10 months by the Stanford-Binet. She cried in the weak tones of a small baby when I took her from the hospital and brought her to the laboratory, and she again cried when I took her back. She could walk, but would only sit on the floor and play with colored sticks. There was something about her behavior which seemed really uncanny. She would wave the colored sticks back and forth slowly and watch the end of the stick; then she would pose with one end of the stick resting on the floor. At other times she would sit with the head thrown back looking fixedly at the ceiling and could not be made to change her attitude when called or when

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moved unless one attempted to remove one of her toys. She would do none of the tests which required the slightest amount of cooperation. If not allowed to have her own way, she would scream violently and hit at some one or at herself. Even an imperative tone in one's voice would help to bring on such a spell. She seemed to be a miniature reproduction of adult psychosis which had reached the idiotic stage.

History Since Admission.—Oct. 3, 1918, the patient was taken ill with influenza. Bronchial pneumonia developed a few days later. The temperature increased to 105 F. for several days. This was followed by empyema. The pleural cavity was drained, and a long convalescence followed. During this convalescence, marked symptoms of mental awakening were noted. She began to call the nurses and physicians by name. She distinguished between two picture books, a "Dolly Book" and a "Topsy Book," and asked for the one she wanted. She made a few associations; for example, a nurse whose glasses attracted her, she called "Kelly-glasses." A nurse who had given her some bright hair pins she referred to as "kitchen hair pins." There was a large rabbit on one of the pianos, so each time she heard music she would say "bunny-piano." From this time on, she improved rapidly. Her automatic movements disappeared. She learned to feed herself. She became tidy in her habits, never wet or soiled her clothing, talked in sentences, knew all the letters of the alphabet by sight, buttoned her clothing, and could say her prayers, "Now I lay me," etc. She memorized short rhymes, such as "Mary Had a Little Lamb," and played like a child of 3 or 4 years of age, feeding her doll, dressing and undressing it and putting it to bed, etc. She attended kindergarten and played most of the kindergarten games.

April 16, 1919: The mental age of the patient by the Stanford test was 2.[4] She pointed to her eyes, nose, mouth and hair, named key, watch and pencil. She was very distractible, but was interested for a short period. She learned rather quickly, put knobbed pegs in holes, and on the Witmer cylinder test she improved her method and reduced the number of errors. Exact counts were not taken until about the third trial, as she was playing with these materials while other children were being tested. When observed more closely on the third trial, she put in cylinders with increasing diameters, and those with equal diameters and increasing lengths with as many errors as correct moves, correcting her own errors easily. She sorted three colors of pegs when watched and when her attention was called to error ("No"). She made many errors when not corrected. Her attention wandered. After being corrected she would place the next two colors correctly.

Jan. 18, 1920: The mental age according to the Stanford-Binet test was 3 years, 10 months.

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SUMMARY

This study has aimed to demonstrate the pragmatic principle of studying effects. Instead of learning of effects by studying causes, the pragmatist would learn of causes by studying effects. Hence the fruitful yield of neuropathology which, although studied as effects, has taught us much of causes. Similarly, the study of the effects of influenza may teach us something of the cause of hypophrenia.

The effects of influenza on hypophrenia as presented above may be regarded from a clinical standpoint, in the form presented. Thus, there are cases of hypophrenia aggravated in particular respect to emotional symptoms, to intellectual defects, to volitional disorders; there are further cases illustrative of neurologic, psychopathic and encephalopathic complications (if, indeed, we may regard these as complications).

But studying the problem more abstractly, one may group these effects according to the type of phenomenon, according to the generic process represented. Previous studies [16] have shown that the psychoses subsequent to influenza illustrate in general three processes. These are the processes of creation, precipitation and aggravation. To these there should be added a fourth, the process of amelioration.[36]

I think the phenomena observed in connection with hypophrenia and influenza as illustrated above can be similarly grouped.

The process of creation is not conclusively demonstrated. I have illustrated it imperfectly with two cases, the gross brain lesion and the hydrocephalus developing subsequent to influenza. I do not include here the secondary mental loss accompanying the "dementing" psychoses subsequent to influenza, although it is not at all axiomatic that these *de*-mentias are inherently different from the *a*-mentias of simple hypophrenia. Nevertheless, this point is waived for the time being.

36. This is presented in another article, to be published.

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The process of precipitation is by the very nature of hypophrenia difficult of demonstration. One can conceive of a case of avowed mental incompetence, whose frank defectiveness appeared suddenly only after influenza. But such cases have not been reported, or observed by myself.

But the process of aggravation, the intensification or augmentation of the symptoms of hypophrenia, is very well illustrated by the majority of the cases presented above. One might conclude from this small series of cases, which, small as it is, represents perhaps the largest collection of cases dealing with this feature of somatic-psychiatric interrelations, that aggravation is the chief process represented by the interaction of influenza on hypophrenia, where any effect at all is perceptible.

This is in striking contrast to the observations made in the cases of postinfluenzal psychoses. Here it was found that creation and precipitation were the processes primarily represented, and augmentation was comparatively infrequent. To be sure, I did not carry on as extensive an inquiry by questionnaires as to the effects of influenza on patients already psychotic; but this feature of the study was not entirely neglected, and as far as carried it yielded negative results.

Finally, the process of amelioration can be included, as illustrated by one very remarkable case. Amelioration of mental affliction *ex nocentibus,* i. e., by injurious agents, is, as I have said, not confined to influenza as an agent or hypophrenia as material; but whatever its mechanism, it is unfortunately extremely rare. Some day it may suggest some new line of attack which will yield rich fruit.

CONCLUSIONS

1. The awakened interest in the study of the effects of hypophrenia (feeblemindedness), together with the advances made in neuropathology, have put us in a receptive attitude toward the theories of exophysical

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causation of feeblemindedness, and have stimulated constructive research.

2. The pragmatic value of studying effects rather than causes was overlooked by the older writers, who, taking for the most part the standpoint that hypophrenics are "born, not made," persist in it inflexibly in spite of clinical evidence and statistics which would indicate the importance of the effects of infections and the possible infectious causes of feeblemindedness.

3. Influenza as a type of acute infection, with known neurotoxic potency, gave opportunity for study of its effect in relation to hypophrenia in the recent epidemic.

4. Inquiries addressed to superintendents of state hospitals for the feebleminded afforded seventeen replies, representing more than 16,000 patients, but were almost entirely barren of important results, owing in part possibly to the burden and pressure of duties incident to the care and alleviation of the physical disease.

5. This negative evidence is offset by data from the cases of our Boston series and from private practice, illustrating effects of influenza on hypophrenia.

6. The uncomplicated cases represent: (1) hypophrenia (feeblemindedness) with aggravation of intellectual lack prominent after influenza; (2) hypophrenia with emotional sphere disturbances notably intensified after influenza; and (3) hypophrenia with volitional and conduct disorders conspicuously aggravated by influenza; to which may be added (4) a single instance of symptomatic improvement, in all spheres but notably that of intellect, following influenza. (*Vide infra.*)

7. The complicated cases represent: (5) hypophrenia with conspicuous neurologic manifestations after influenza; (6) hypophrenia with psychotic manifestations following influenza; and (7) hypophrenia with psychopathic manifestations prominent after influenza.

(These seven groups may be tersely summarized,

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using Southard's terminology, as hypophrenia with hypognosia, hypophrenia with dysthymia, hypophrenia with parabulia, hypophrenia with improvement, hypophrenia with encephalopathy, hypophrenia with psychosis, and hypophrenia with psychopathy. In each case it is understood that influenza was the apparent effector.)

8. Generic processes illustrated as to the rôle of the influenza are comparable to those observed in the study of the psychoses associated with influenza, namely, creation, precipitation, aggravation and amelioration.

9. The process of creation is very imperfectly illustrated by two cases, both open to question.

10. The process of precipitation is not illustrated at all; no cases were observed, although the possibility is not inconceivable.

11. The process of aggravation, however, was comparatively frequent, and is illustrated by the majority of the cases here cited.

12. Amelioration is illustrated by one case, by courtesy of Dr. W. E. Fernald.

13. In this, then, the relation of influenza to hypophrenia differs from that of influenza to the psychoses, since aggravation in the latter was the least frequently observed phenomenon, and in the case of hypophrenia the most frequently observed.

14. From which we conclude that:

(*a*) The usual effect of influenza on the brain is not the production or precipitation of hypophrenia, and if it ever produces hypophrenia it is probably by means of a more or less obvious encephalopathy.

(*b*) On the other hand, of those already manifesting mental lack, certainly a few are influenced adversely by influenza, and the symptoms of hypophrenia aggravated, an aggravation which may be predominantly in the intellectual sphere, in the emotional sphere or in the volitional sphere.

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(*c*) Psychoses of an indeterminate type are occasionally precipitated by influenza in the feebleminded, even as in normal persons.

(*d*) At least occasionally, though rarely, the effect of influenza on hypophrenia may be symptomatic amelioration.

Mulvane Building.

ABSTRACT OF DISCUSSION

Dr. Andrew L. Skoog, Kansas City, Mo.: Dr. Menninger laid stress on the possibility of influenza affecting the somatic as well as the psychiatric disorders of the nervous system. He stated in a general way that influenza may precipitate feeblemindedness, but that most of the feebleminded states are merely aggravated by the added acute illness. The case of simplemindedness cited illustrating the possibility of relative or symptomatic improvement as the result of influenza, is very interesting. In certain other diseases, notably in epilepsy, also mentioned by Dr. Menninger, I have seen improvement occur following acute infectious diseases. Why that is the case, I do not know. It is possible that in feeblemindedness, where a toxin or an organism is affecting the nervous system, development or growth is stimulated by irritation of the neurons.

Dr. Hyman Climenko, New York: During the last two epidemics of influenza, lethargic encephalitis either ran parallel with or immediately followed influenza. It would, therefore, be very important to rule out encephalitis in the cases reported by Dr. Menninger. In one group of his cases, the encephalopathies, some very suggestive signs of encephalitis are in evidence. The improvement in these cases strongly suggests lethargic encephalitis. Was a lumbar puncture made in all these cases? What were the findings in examination of the cerebrospinal fluid?

Dr. Oscar J. Raeder, Boston: The case of feeblemindedness in which improvement took place in the mental condition, following an attack of influenza, is a most interesting one. I can understand how a toxemia would cause an augmentation of hypophrenia, but this case brings up a new point. I know of a similar case of feeblemindedness in which the patient had an infectious disease followed by pneumonia and empyema. During this illness the patient had a very high temperature but made a complete recovery. Following convalescence, the child showed marked improvement of the hypophrenia evidence. The improvement in these cases strongly suggests intelligence quotient. The question presents itself rather as to whether this might be the awakening of some sort of endocrine hibernation. Perhaps, the stimulation resulting from the toxemia or high temperature caused the arrested develop-

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ment to be pushed over the bar, so to speak, and improvement in the physical condition of the patient and raising of the intelligence was the result.

Dr. Tom A. Williams, Washington, D. C.: In my opinion, Dr. Menninger's theory can be expressed much more simply in this way: We all know that the influenza poison has a most deleterious effect on the whole organism, including depression of the cerebral function. Nearly all of us who have had a severe attack of influenza have experienced, sometimes for a long time, periods of greatly reduced psychic potentiality; we carry on with impaired intellectual functions; but we carry on, nevertheless. The disturbance frequently takes the form of depression. In a case of hypophrenia psychometric methods will show definite lowering of function, as expressed by the hypophrenic and other tests. It can scarcely be expressed in measures by those who have a higher plane of intelligence. Therefore, what appears to be an aggravation of feeblemindedness is, in reality, only depression of cerebral function. Before one could say that there is an increase in the feeblemindedness, these patients must be observed over a long period, a year at least, because experience has shown that in the majority of cases of postinfluenzal complications recovery ensues after some months. It would not seem, therefore, that we are dealing with a condition very different from the familiar toxi-infectious psychic states which occur after infectious diseases, especially influenza.

Dr. C. F. New, Indianapolis: Do you regard these reactions as characteristic of influenza in itself, or are we not apt to get similar reactions in these persons when they contract infectious diseases from other causes?

Dr. Karl A. Menninger, Topeka, Kan.: Routine lumbar punctures were made in all cases, together with complete physical and mental examinations, all of which are included in the original data. Syphilis was definitely ruled out. Answering the second query: I think influenza itself is not so important, except that it has given us an opportunity to study somatic and psychic interrelationships. We do not know much of this part of psychiatry. As a matter of fact, no book in the English language deals with the mental complications of influenza. Anything which will contribute to our knowledge of somatopsychic interrelationships ought to be favored. Typhoid fever is rapidly disappearing; therefore, further study of that disease will soon be impossible. Influenza offers that opportunity and we should take advantage of it. Dr. Williams suggested that what I have said could be simplified. I think so and I hope so. I want to further the minute analysis, the critical study, of the data presented, right now while they are available. I hope that eventually the results will be shaped into more generic principles as Dr. Williams has intimated that they can be.

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OUTLINE OF A SCHEME FOR WRITING THE NATURAL HISTORY OF SYPHILIS

SANGER BROWN, M.D.
CHICAGO

Of the diseases that affect the human race, syphilis is certainly among the most formidable; and while within the last decade or two, owing to the brilliant development of laboratory craft, our conceptions of the malady have been materially extended and clarified, no corresponding progress has been demonstrated in the matter of prevention and cure.

About fifty years ago, before the natural history of pneumonia had been established, the scientific programs of medical society meetings, and the pages of medical journals presented a liberal display of papers extolling the specific virtues of various remedies in the treatment of that disease. When its natural history was understood, however, it immediately became obvious that nearly all the advocated measures of treatment had been more or less harmful and, indeed, in not a few instances had directly contributed to a fatal result. While the revelation of its natural history has not so far led to the discovery of a cure for pneumonia, it certainly cleared the field for action and has mitigated much suffering and saved many lives. If the natural history of syphilis were known, advantages might be expected similar to those referred to in regard to pneumonia.

In the treatment of any disease, the practitioner's ability to assess correctly the value of the remedies employed must depend on his knowledge of its natural history. It is recognition of the importance of this

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maxim that has prompted me to propose a plan for writing the natural history of syphilis; for so far this has not been satisfactorily accomplished, nor at the present time is there anywhere evidence of a movement in this direction.

Two outstanding obstacles bar the way when the task of ascertaining the natural history of syphilis is contemplated: One of these is created by the social and moral stigmas with which, according to popular verdict, syphilis stamps its victims. The other owes its existence to the protracted duration of the disease. Without my attempting either to state or to discuss the different views entertained at present regarding the course and curability of syphilis, probably all will agree that, in order to obtain the most accurate possible knowledge of the natural history of the disease, critical observations should cover the entire lifetime of a great number of patients, from birth when possible, but in any event beginning with the primary infection.

That the necessary histories can be secured and profitably studied only by an earnest and sustained coordinated effort of our profession may appear more obvious if some of the conditions incidental to the work are briefly stated.

NECESSITY FOR COOPERATION

In a considerable proportion of cases, a syphilitic patient, whether treated or not, regains his usual health within a period of two or three months of his primary infection, and even though during this period adequate clinical notes had been made, in many instances and for various reasons the physician is afforded no further opportunity of amplifying his record. Not until twenty years later, perhaps, when symptoms of tabes or paresis appear, is the medical history resumed. Assuming that the clinical notations cover the remainder of the patient's life, including a necropsy, and that the professional services throughout were of the highest order, then so far as supplying a valuable contribu-

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tion to the study of the natural history of syphilis is concerned, the commendable efforts of the several physicians connected with the case are largely wasted, because for a considerable period the patient would not have been under medical observation at all, and because it would rarely happen that the notes covering the period of initial infection would be available to the physician in charge during the final stages. Incomplete histories would be of only limited value for the purpose in view. In order to carry the proposed investigation to successful accomplishment many hundreds, perhaps thousands, of complete histories must be available for competent analysis. Manifestly these can be accumulated only by some efficient scheme of cooperation.

For the purpose of enlisting and maintaining the interest and cooperation of the patient, appropriate propaganda should be prepared. This should appeal to his altruism; should convince him that his own interests would be best conserved by continuing for the remainder of his life under close medical supervision; should assure him that his acquiescence would in no way compromise his rights of professional privacy, and should make it clear that thenceforward without incurring additional financial obligation he would receive the highest grade of medical service anywhere obtainable.

While many patients for various reasons would undoubtedly decline the terms submitted, it does not seem improbable that a sufficient number might accept and adhere to them to meet the requirements of the proposed investigation.

Conceding that it is important to obtain the specified histories; that a sufficient number of physicians might be induced to volunteer their services for the work incident to their production, and that the necessary cooperation of patients could be secured, practical plans for the successful prosecution of the project present themselves for consideration.

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PROPOSED COMMITTEE AND ITS FUNCTIONS

The investigation of a subject like this one, which challenges the serious concern not only of the nation but also of the whole human race, and which for its succcessful prosecution requires the coordinated effort of a large number of well qualified physicians, should obviously be conducted under the auspices of the American Medical Association, our strongest, most representative and most authoritative medical organization. The appointment of a standing committee for this purpose would in my opinion constitute a legitimate expansion of the activities of the Association.

Assuming that a committee had been formed as suggested and was prepared to proceed with the work, meetings would have to be held to determine its scope, and methods to be adopted in carrying it forward. A campaign might be conducted to enlist the interest both of the laity and of the profession. Appropriate papers might be prepared and presented where they would be most effective. The services of both the lay and the medical press might be sought. The profession might be canvassed for volunteers who would be willing to contribute their services to the actual work of taking histories according to the prescribed methods. A manual setting forth the most approved and serviceable information on the subject of syphilis might be compiled for the instruction and guidance of workers.

In anticipation of flagging interest, the committee might see to it that close relations with the volunteers were maintained so that timely assistance and encouragement could be given. This would involve considerable correspondence and possibly quite numerous personal interviews, lectures and demonstrations. In the event of interruption of the relations between an observer and a patient, a method could be devised to secure continuation of the clinical record. Provisions would have to be made for analyzing, classifying and filing the histories, which would require both medical and clerical service.

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The foregoing outline of the duties which would be required of the proposed committee demonstrates that, from the first, funds would be needed, and that it would be idle to attempt to carry the work to a successful issue without the assurance that ample funds were available.

CARRYING OUT THE PLAN

For the purpose of contributing to a more definite understanding of the proposed plan, let it be assumed that all desired conditions had been met. Then in some populous center, a constituent society of the American Medical Association, in which to initiate and develop methods for taking, collecting and filing the specified histories, should be selected. Those willing to participate actively in the work would have to organize; and from the first, to insure progress, the organization would require the services of a qualified paid executive. It is quite possible that the advisability might become apparent, as the work proceeded, of providing hospital accommodations for a limited number of patients presenting symptoms demanding special facilities for their satisfactory observation. As experience ripened, units might of course be multiplied as desired. Several units would naturally suggest the necessity of a central bureau from which all authority must emanate and to which a report of all activities must be submitted as the central committee might direct.

The scheme certainly sets a formidable task. It offers neither individual fame nor pecuniary reward; and while some beneficial results, especially from the educational side, might be expected in the first decade, fifty years would probably be required for completion of the investigation, so that few if any of the earliest workers could hope to witness the full results to which their efforts had contributed. A strong spirit of altruism, high courage and energy are demanded for the initiation and prosecution of the subject. Hesitancy, criticism and excuses are only natural under the circumstances; and yet, from the clinical side, unless we

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accept present methods of study and practice as final, a critical survey of the situation discloses no satisfactory alternative.

It may be stated here that a large standing army, providing its permanence could be secured, presents some exceptionally favorable conditions for study along the lines suggested. Some valuable results of work done in the Austrian army have already been published. But at best the range of military practice, under ordinary conditions, is not comprehensive enough to meet all the requirements of the proposed investigation; and furthermore, it does not include the highly important educational features to which reference has been made.

QUESTIONS THAT WOULD BE ANSWERED

In my opinion, from the clinical side the submitted scheme more than any other promises to shed needed light on such questions as:

1. What are the immediate and remote effects of various methods of treatment?
2. What proportion of syphilitics develop paresis or tabes?
3. Does any method of treatment influence liability to paresis or tabes?
4. In what proportion of cases does the Wassermann test remain positive in spite of all methods of treatment?
5. What are the various relations between cerebrospinal fluid and blood Wassermann findings? From a study of these can liability to paresis or tabes be predicated?
6. Can the wife of a syphilitic patient who is symptom free but has a positive Wassermann test acquire a positive Wassermann test from her husband without ever manifesting any symptoms of infection? And if so is she immune from further reinfection? And what is her liability to paresis and tabes? Similar questions would, of course, apply to the children of syphilitics.

This list of questions is intended to be merely illustrative; the investigation should undoubtedly cover the whole range of syphilitic pathology, and if the suggested committtee be appointed, this should be duly recognized in its membership.

I respectfully submit that none of these or similar questions can be answered satisfactorily until the natural history of syphilis has been written.

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PROPOSED RESOLUTIONS

I submit the following preambles and resolutions for your consideration:

WHEREAS, The deleterious effects of syphilis on the mortality and morbidity of the human race are so prevalent and so severe as to challenge the most serious attention of the entire medical profession; and

WHEREAS, In the scientific study of any disease, knowledge of its natural history is an item of cardinal importance; and

WHEREAS, Owing to the protracted course of syphilis, a continuous and complete clinical record of a given case can be secured only through the services of several successive medical observers; and

WHEREAS, It is highly desirable that a sufficient number of completed histories be accumulated and preserved, and made easily accessible to students; and

WHEREAS, For the successful accomplishment of the purpose set forth above, the interest and cooperation of a considerable number of the best elements of our profession as represented in the membership of the American Medical Association are necessary; therefore be it

Resolved, (1) That the Section on Nervous and Mental Diseases of the American Medical Association recognizes the importance of ascertaining the natural history of syphilis and of making this history accessible and in form serviceable to students of medicine; further,

Resolved, (2) That the Section on Nervous and Mental Diseases of the American Medical Association respectfully requests the trustees of the American Medical Association to appoint a committee from the sections most immediately concerned, whose duty it shall be to devise practical means and methods of accomplishing the foregoing specified purpose; and further,

Resolved, (3) That the representatives of this section in the House of Delegates be requested to present these preambles and resolutions to the House of Delegates, and to ask its endorsement.

59 East Madison Street.

ABSTRACT OF DISCUSSION

DR. HUGH T. PATRICK, Chicago: No one could question the wisdom of this matter if it could be carried out. We all agree that it is something devoutly to be wished for. The difficulties lie in the details of carrying out the plan, but if they can be worked out by a committee so as to be made practical, as indicated by Dr. Brown, we have reason to believe that the necessary funds can be procured. Papers have been read to show that the incidence of tabes and paresis has no relation what ever to the previous treatment for syphilis; that tabes and paresis occur just as frequently in patients who have previously received good treatment for syphilis as in those who have not; and papers have been published to prove just the contrary.

DR. W. S. LINDSAY, Topeka, Kan.: Twenty years ago I treated a young man who had syphilis with the old-time salvarsan. About ten years ago several Wassermann tests were made, but the results were negative. He came to me for certification for marriage. On the basis of these negative reports, I signed his certificate. He has continued to have negative Wassermanns, but the woman he married, whom I knew in childhood, as I had known him, had a stillborn child

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and after that a positive Wassermann. She had a second stillborn child and continues to have a positive Wassermann in spite of arsphenamin treatment.

Dr. Charles D. Humes, Indianapolis: I have been confused with the utter lack of knowledge regarding the date of infection, the amount of treatment that has been given, the nature of the after-care, if any, the character of the medical treatment, and the medical status of the patient immediately preceding the neurologic break. If those factors could be eliminated it would be a tremendous help to us and no doubt save many patients from the serious after-effects of syphilis. But until we can remove all the elements of human frailty and commercialism, it is very difficult to get patients to return to the office for so-called free consultation.

Dr. C. R. Woodson, St. Joseph, Mo.: I am heartily in favor of the resolutions that have been presented. The carrying and resisting capacity of men varies greatly, and it is hard to tell who will and who will not have paresis or tabes. I believe that paresis is less frequently found in those who have had thorough treatment than in those who have not had such treatment, but more particularly in those who have all "plus" habits, rather than in those who have excellent habits. A pathologist of some experience has expressed the belief that paresis was extremely rare, and perhaps never existed in those men who have never dissipated. I do not remember having observed this but if so it is in line with my belief of the effect of the "plus" habits.

Dr. Sanger Brown, Chicago: This paper was not intended to bring up any discussion on the course of syphilis but simply to outline a plan which might enable us to secure reliable data to work with when we wish to establish the value of any particular method of treatment that might be employed in the disease. Before the natural history of pneumonia was understood, a considerable part of the programs of medical meetings was taken up with papers extolling the specific virtues of certain remedies in the treatment of that disease. In the past few years the programs of medical meetings have shown a similar display in regard to syphilis. After the natural history of pneumonia was demonstrated and understood, the papers ceased, and it was understood that the extolled specific remedies had been, for the most part, meddlesome and harmful and in some cases directly contributory to a fatal result. It is quite possible that when the natural history of syphilis is understood, similar results may be noted.

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THERAPEUTIC AGENTS IN CHRONIC NERVOUS DISEASES *

W. A. JONES, M.D.
MINNEAPOLIS

Chronic nervous diseases include the various types of mental and nervous diseases, defects, and associated illnesses that are interrelated with the somatic system. As approximately 50 per cent. of all diseases, of whatever nature, are primarily or fundamentally due to mental influence in its broadest sense, we have a basis for methods of treatment. The more definitely limited disorders of the nervous system not controlled by mental states are comparatively small in their inception. This class may include defects in the structural pathways, and if diseased, they are not uncommonly precipitated by emphasized injuries, shocks or infections.

The gradation between organic nervous diseases and diseases of nonnervous tissue is frequently difficult to analyze and differentiate; hence we are led to assume that a complication of chemical and physiologic states exists, rendering a final diagnosis speculative or tentative. We now know that a thorough history and an elaborate physical investigation are essential before a conclusion is reached, as both the history and the examination usually lead us in the diagnostic path, or, at least, are suggestive and therefore of diagnostic value.

As we study our nervous problems we necessarily become convinced that defects in one or more systems are so closely interwoven that a syndrome of symptoms frequently provides a screen behind which we

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are unable to go in our efforts to outline a definite scientific diagnosis or prognosis; hence we emerge from our consultation room with a dubious mind. We are almost daily confronted with new methods of investigation and research that require the assistance of the internist, or the specialist, with his modern apparatus and his painstaking efforts to exclude or include his theoretical findings. The surgeon, with his mind bent on surgical prospects, strives to impress us with his surgical diagnostic knowledge, in order to convince both the neurologist and the internist that, fundamentally, their combined explanations are insufficient. And he may attempt to draw from the neurologist's and the internist's intensive studies immaterial and irrelevant conclusions because a pathologic process is demonstrable.

The endocrinologist is temporarily in the foreground, and we gasp and speculate as to whether or not he is on the right track. He has so many theories to offer and so many deductions to declare that he sometimes obscures a possible diagnosis; or he may clear up some of our doubts by his investigation of ductless glands and his applications of remedies that may be of great value.

Then, too, the bacteriologist and the serologist confuse us greatly; they know we are all wrong and are willing to admit that they are right, and we bow our bewildered heads before the laboratory phenomena. From their intensive studies, we are sometimes almost persuaded to admit that nearly every one may have a little syphilis in concealed areas, on the assumption that this disease has been promulgated through the centuries, yet remains in retirement for the reason that no positive evidences are presented to substantiate this view.

Considering all these and other unmentioned possibilities of arriving at a tangible or tentative diagnosis, it seems permissible to conclude that the human

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race is constructed of poor materials, put together in a disorderly and disorganizable mass. The patient, or the case, may be interesting from many points of view; but the individual who presents a multiplicity and a complexity of problems, together with his variegated life, his conglomerate up-bringing, his environment, his occupation and his social status, is really the one for our first consideration. It is not astounding that he baffles us, and tempts us to treat him for what he is not. In spite of all these mishaps, the psychiatrist and the neurologist have demonstrated their worth and distinction, based on the accomplishments and the elevation of their art in the past three or four years. And they seem destined, ultimately, to occupy a higher plane in medicine than has been their lot heretofore. The field of the psychoneurologist has been for many years the dumping-ground for undiagnosed disorders, but today he is the watcher of the melting-pot of diseases. With all of these recognized distinctions that have been showered on him, does he actually know how to treat his psychoneurotic patients; and why does he so persistently cling to the old methods of treatment, unless it be that he admits that the majority of his patients are studies almost beyond his comprehension?

Among the many modes of treatment that have been suggested to us, rest seems to be of uppermost importance. It is common knowledge that many of our neurologic and psychiatric people are told that they are simply nervous and that what they need is a life filled with interesting and diverting occupations. A number of them are sent on long and arduous trips in the belief that travel and change of scene, and life in the open air, carried on in a strenuous manner, constitutes the most highly beneficial treatment that can be employed. Of these, many come back to occupy the same place they held before this unwarranted method was carried out. It is quite true that

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in a few instances a strenuous life, provided it is well regulated and well ordered, among the types who are sufferers from nervous irritability, does help them remarkably. An investigation of their former life shows they were sedentary and that their minds were glued to routine channels and their bodies to an office chair. This type of man has a one-track mind devoted only to business, to which he applies himself overassiduously; and he provides no escape for his dissipated energies. He becomes stagnant and stale, physically, nervously and mentally. Put him under proper muscular discipline, regulate his habits, and sweat out the bankrupt skin and flabby muscle accumulations, and clean out his gastro-intestinal tract by normal physiologic methods, and he comes out clean, wholesome and well. These cases, of course, must be carefully selected; but a large number of patients of this type are congenitally or constitutionally inferior, and need a prolonged rest in a suitable environment. The tendency of the day is to have these people rest for a brief period and then get them up and push them about. This can be accomplished only by a skilled attendant, and often the result is lamentable. Give them a longer rest period, add to their diet, and see that they are rested, not only nervously but mentally. Then, through a gradual process of exercise and outdoor life, they come back by degrees to a condition approaching a normal state. It is among this class that we get those who have never been instructed as to the care of themselves; who know, indeed, nothing about ordinary health measures. They become blockaded in their muscular, arterial and glandular systems. It is among these people, too, that many cases of chronic invalidism are found, mainly because their condition was not recognized early and they were not put in bed with a definite object in mind. We see, too, many instances of profound depressive states where a

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timely suggestion would put them to rights. And these people, too, instead of being worked inordinately, physically and mentally, are the people who need a long, peaceful and well-ordered rest in bed.

The developing dementia praecox patient, or the constitutionally inferior individual who has been crowding his life with all sorts of work and pleasure, with irregular hours and without sufficient rest, is the one who very soon shows his limitations, and his reconstruction requires excessive patience and a carefully directed line of treatment before he shows a prospect of improvement. Unfortunately, among this class are the people of comparatively small means, or those who, perhaps, have dissipated their finances as well as their physical strength, and who are unable to carry out a comprehensive or a well-adjusted scheme of living. We may, in many instances, be obliged to resort to halfway measures in order to meet the circumstances of the individual, and not infrequently they can be restored to a fairly balanced life and permitted to engage in occupations less strenuous.

Exercise, in its various phases, is often carried too far, because the life of the individual has not been accurately analyzed and his exercise suited to his capacity. Sending a mentally defective person to a gymnasium with some carelessly offered suggestions as to exercise is to be deprecated. Frequently the overexercise of people who have just recovered from minor or grave infectious disorders, and who have an inherent constitutional defect, is detrimental rather than beneficial, and the exercise becomes a dangerous punishment rather than a relief.

The question of foods is more important than we are accustomed to believe. One rule holds good, however—that is, before a process of diet is inaugurated, the patient should be cleaned up inside and outside as well, and then it should be the function of the physician to put the gastro-intestinal tract into such

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order as will permit the individual gradually (and sometimes rapidly) to accustom himself to eating anything that is within reason. Too careful dieting of the average man is a waste of time. Almost any patient can be fed, and even forcibly fed (that is, be urged to eat) anything and everything that is good for him; while another man who goes about with a moderately poor diet list and a book on calories under his arm, and a fear and apprehension of food in his head, not only suffers himself, but brings suffering on his immediate associates.

Baths are much overrated, unless they are applied for some specific purpose, such as a prolonged bath for an acutely excited patient. It may be said, in passing, that the average person does not know how to keep himself clean, and therefore a regulation of his bath toilet is not only beneficial from the point of cleanliness, but serves to improve his skin and muscle activity. The ordinary warm bath and cool spray and the Scottish douche are applicable to many, and they are among our best therapeutic agents. When we send our patients to resorts for baths we are simply transferring the individual from one climate to another, and perhaps by force of example he bathes, as he should, more often than is his custom. We get him out of his old environment, he is taught how to employ his time, and he looks forward to his common, daily, ordinary bath with pleasure because it produces comfort and ease. But most baths given in a routine manner, as is generally practiced at bath places, are more or less valueless. They are on a par with the drinking of so-called mineral water and salt water; the beneficial result consists mainly in the larger intake of water than is practiced at home.

Massage and its adjuncts are fads in the majority of instances, or they are ordered simply to employ and take up the time of the person, who, when he reaches a certain point, has little or nothing to do or who

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declines to take a normal amount of physical exercise. The average man who practices massage as a profession is not a very great help to the neurologist. He may be trained in his specialty, but he is not trained in his mind, and he assumes that massage is the one and only factor in the treatment of disease, just as the new schools of therapy (the osteopathic and the chiropractic) claim to accomplish marvelous results irrespective of other methods of treatment. It is undoubtedly true that these so-called practitioners do much good, because they have the knack of inspiring the patient with the wonderful results of their treatment. And yet, who has not seen the disastrous results following the indiscriminate prescribing of massage in any of its forms?

Following this line are the newer cults who practice strange and inexplicable forms of healing. They employ suggestions, but not the suggestion based on the scientific method of the neurologist. Their system of analysis is inadequate, to say nothing of being unscientific. Their methods of treatment are bolstered up by all-impressive notoriety backed by the newspapers and heralded from mouth to mouth by the credulous. Occasionally a medical man sees the application of suggestion and healing by his impressiveness; and he seeks a wild and remote spot and from this point he permits to leak out the fact that he is a wonder. Within a remarkably short period of time the defectives and the chronically, constitutionally inferior people hear, with widely open ears, of his wonderful cures. It is related that one man in an Eastern state, after graduating from a well-known Eastern university with a full-fledged degree of M.D., established a few barrels of concoctions, gave his awestruck patients a rapid ocular examination, and sold them a bottle of medicine for a price that would be acceptable as an ordinary fee for the average medical man. He finally grew tired of attempting in any way to analyze his patients, and

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simply gave them a rapid glance, followed by the well-known bottle. Eventually the crowds became so great that he issued "privilege" permits (for a substantial fee), in order that the patient should not be too long detained in the acquiring of his bottle of water and herbs. Other healers have profited by the credulity of the public and have practiced their unholy schemes with financial benefit. Now the country is overwhelmed by the so-called "Divine Healer," who admits that Christ practiced the healing art and produced remarkable cures, but that for nearly 2,000 years the art was lost until it appeared in him while yet a child. He goes from city to city, and, with the aid of a devoted churchman, he fills the churches with the lame, the halt and the blind, and, offering a prayer, he lays his hands on these poor derelicts. He heals them without money and without price, accepting what is donated by his grateful recipients; and it is said, without fear of contradiction, that he makes a very good living.

When we come to the final analysis of the whole situation, and the treatment of chronic nervous diseases, the personal influence of the man in charge has much to do with improvement and recovery, provided he is endowed with the qualities of a disciplinarian and the courage of his convictions after he has made a thorough and careful examination and has outlined a course of treatment that is right for the individual. The mere fact that people have faith in their physicians is confirmed all over the world; and when they are presented with a proposition put in a plain, common-sense and direct way, the cures of their physician far exceed all of the cures that are effected by the faker and the charlatan. But the physician's inordinate modesty and his ethical sense prevent him from acquiring a widespread influence which might be of great benefit to others who know nothing of him, his methods, and the application of the scientific mind to the treatment of disease.

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SUMMARY

If it were within the power of the neurologist to treat the rich as well as the ordinary classes, they should be given the same strenuous treatment. This perhaps will not appeal to physicians generally, as it would mean a vast reduction in their fees. But, taking the majority of patients who consult the neurologist and psychiatrist, isolation from their former surroundings, confinement in a well-ordered hospital, and the prohibition of visitors should be rigidly adhered to. Nervous and mental patients do better when they have been regulated by correction of their physical disabilities and forced to follow a definite mental disciplinary line of treatment. This includes all that may be said of enforced rest, not for weeks, necessarily, but perhaps for months. These people come to us depleted in their physical and nervous forces, and a period of rest is needed to balance the years of bad living that bring them to the house of the physician. The establishment of order by discipline applies equally well to the physical as to the mental attitude of the patient, and this can be done successfully only by one who is trained for this work. It is better to make a few mistakes through overdoing this method of treatment than to make many mistakes by underdoing it.

Baths of simple type—warm tub, cool spray or Scottish douche—are of more value than many drugs.

Exercise of the graduated type during the convalescent period is of tremendous value, provided it is carried over many days and weeks and with but moderate increase in the time given to such exercise.

Sufficient examination should be made from time to time to assure the physician, as well as the patient, that nothing important is overlooked. The ordinary routine visits must be suspended at intervals to make this impression lasting.

One person should be responsible for the patient, and only minor details should be left to subordinates.

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The patient should be left alone, and without a special nurse, as soon as possible; otherwise he becomes dependent on the nurse not only for his physical but for his mental habits.

It has been found that massage is not absolutely essential either to the patient's comfort or to his improvement—that his state of mind and his attitude toward himself and others evoke sufficient mental activity to keep him physically fit.

The coddling of patients should be eliminated. Giving way to the wishes of the patient, when we know that doing so is detrimental to his progress, is a backward or retrogressive step. They should all be held firmly in hand by the personal influence of the attending physician, and this rule should not be relaxed until convalescence is well established.

Occupation, unless it is carried on by a trained and skilled worker, is of very little value, and means only the use of time which might be employed in a more profitable manner.

Many patients require a firm, guiding hand. Others require the influence of a strong and dominating will, together with mechanical or drug restraint, and confinement in a suitable room. If this method of treatment is begun early in selected cases, the recovery is much more rapid and satisfactory.

ABSTRACT OF DISCUSSION

Dr. Frank R. Fry, St. Louis: The neurologist must support himself by reliable internists. With data thus obtained he can decide the physical and the psychic side of each situation that he is handling. By checking up our cases carefully from the physical side, we can more readily determine how to handle them from the psychic side. Compared with this the technic of a rest cure is of secondary importance.

Dr. Ross Moore, Los Angeles: The suggestion of not having a special nurse is a very important one: not allowing the patient to be treated, but to have his mind focused on what he himself has to do in order to get well. I do not like massage, except when a patient is so physically run down that he needs it for physical reasons. Patients get

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the idea that massage or hydrotherapy, or what not, will make them well. Along the same line is the question of relaxation.

Dr. C. R. Woodson, St. Joseph, Mo.: It is hard to lay down a set of rules by which to treat nervous men and women. Rest is excellent in the treatment of some patients and is desirable. In some cases continuous rest is not desirable. In some cases it is impossible for a time to get rest and no one has ever shown us how to get it. A business man who has broken down and is badly depressed must be taken care of and exercised by an entertaining nurse, one who can divert the patient. A maniacal, badly depressed patient, with elevation of temperature, acceleration of heart action and in a constant state of muscular and mental agitation must be looked after constantly. Feeding is necessary. For a patient who will not eat regularly, tube feeding is necessary. If a patient who is badly depressed is left alone, he will kill himself, and this is not to be desired. A maniacal patient should not be left alone, as he will bruise himself on the walls or bed, will expose himself by getting the covers off his bed or getting on the floor and will contract pneumonia. As to filling a patient with drugs, no neurologist or internist will want to do this, as it will not have the desired effect. It is impossible to lay down a set of rules for the treatment of insane patients. Persons who suffer from a mild neurosis may be treated as Dr. Jones suggests. A person who can divert a melancholia patient and who can get his mind off of himself, who will go walking with him, will be good for him. If you lock the melancholia patient up and tell him he must rest, he will not be satisfied. If you shut out his friends he will not be satisfied.

Dr. H. Climenko, New York: One form of psychoneurosis has baffled me. This is the anxiety neuroses with marked vasomotor symptoms following a psychic trauma. This neurosis occurs usually in middle aged people, both men and women. The patient is really not insane. He cannot adjust himself to his environment. These people are always brooding over the wrongs done to them. Every time they think of the psychic trauma they become covered with a cold perspiration, develop a rapid pulse, and frequently irregularities of the pulse due to extrasystoles may be present. I have succeeded in curing a small number of these patients by introducing new interests in life. These interests were always of an ideal character. The large majority of these people were not amenable to any known form of treatment.

Dr. W. T. Williamson, Portland, Ore.: The point at issue is simply the question as to the particular quality and character of the patient. One individual requires a certain form of treatment, while another person with exactly the

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same trouble will require a method of treatment which is exactly the reverse of that employed in the former case. Rest is, perhaps, most generally necessary for those whose nervous system is run down, and very often it is required that this rest be prolonged, the patient carefully watched and treatment carried to the degree set forth by Dr. Jones. Experience has shown, I believe, that rest, properly watched, is the most helpful form of treatment for that class of people called neurasthenics, and that extremes of treatment should be employed only in those cases where we have more definite psychic derangements. Rest carried to the extreme, which was opposed by Dr. Woodson, would, of course, be wrong. Rest can be abused.

If a patient who requires watchfulness to protect him be deprived of a special nurse while having that rest, the results of rest would not only be nullified, but the patient himself would be exposed to additional dangers. We must not have a general system of treatment for any disease. The physician who treats a disease on the basis of its name is bound to blunder. We must simply take the individual as we find him, forget for a moment the name of the disease from which he is suffering, and treat him according to the conditions found, not following any stereotyped methods.

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CONGENITAL FACIAL PARALYSIS

TWO ADDITIONAL CASES

FRANK R. FRY, A.M., M.D.
ST. LOUIS

Dr. Michael Kasak and I [1] last year presented to the American Neurological Association the notes of a case of congenital facial paralysis of unusual interest. Two additional cases I believe deserve to be recorded, and I am therefore presenting them on this occasion. As a rather extensive bibliography accompanied our paper last year, I am omitting it here.

REPORT OF CASES

CASE 1.—Helen B., aged 1 month, was first brought to the pediatric service of the outpatient department of Washington University School of Medicine, Sept. 9, 1916. On physical examination it was evident that motility was absent in the lower right side of the face, and the upper portions seemed weak. The lower left side also seemed weak. The lower lip was everted to a considerable degree, and the right corner of the mouth was pulled down more than the left when the patient was crying. The upper lip seemed short and thin. The mother noticed the peculiar condition of the face the next day after birth, and stated that the child was not nursing well on account of it.

September 23, the patient was first examined in the neurologic clinic.

October 11, the child was first brought to the ophthalmic clinic with a slight conjunctivitis, which was improved after several visits.

March 23, 1917, the infant appeared in the otologic clinic. The examination revealed an acute otitis media on both sides, and both membranes were punctured. Within three days the acute symptoms had subsided. The patient was finally discharged from the otologic clinic as well, June 1, 1917.

1. Fry, F. R., and Kasak, Michael: Congenital Facial Paralysis, Arch. Neurol. & Phychiat. 2: 638 (Dec.) 1919.

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Meantime the patient was seen from time to time by Dr. Michael Kasak and others in the neurologic clinic, until the time of her last recorded visit, Nov. 11, 1918. At this time Dr. Kasak and I examined her together. The child was then 27 months old. In repose, the mouth fell open with an eversion of the lower lip. The upper lip seemed relatively short. When crying, the right corner of the mouth drew down, making an unpleasant deformity. This was evidently due to the action of the platysma. The left side of the face also gave the impression of weakness, and especially when the child was crying there was an evident lack of motility in the upper portions of the face as well as in the lower, more so in the right. The mother stated that the eyes did not close completely when the child slept.[2]

In all other respects, the child seems normal. The family history furnishes no items of importance. The labor was spontaneous and easy, and at full term.

This case was in the service of Dr. Sidney I. Schwab, chief of the neurologic clinic. He saw the patient only once, as he was absent in France in war service, during the time of her subsequent visits to the clinic. The child was 6 weeks old at the time of Dr. Schwab's examination. His impression then was that there was a peripheral paralysis of the seventh nerve only on the right side. He kindly accorded us the privilege of using the clinic's notes in our report. Later on, however, the conditions as we have described them were evident, and they recall certain observations of Vogt:[3]

In peripheral facial paralysis the whole side of the face is motionless; in central and in most of the bulbar (types) the superior branch retains its movement. In congenital cases the superior branch remains motionless, while the region about the mouth retains either complete or partial motility. When this difference between the upper and lower part of the face is great, it gives a characteristic facial expression: full, protruding lips which are in contrast with the masklike, expressionless, sunken face above. The appearance of the face makes the condition often recognizable at the time of

2. Prior to this, Dr. Kasak had studied the case carefully, and he and I had expected to include the report of it in our paper on congenital facial paralysis presented to the American Neurological Association, June 16, 1919, but owing to an accident with the notes it was omitted. The credit of its preparation belongs to Dr. Kasak.

3. Vogt, H.: Infantile Beweglichkeitsdefekte im Bereich der Hirnnerven, Handbuch der Neurologie (M. Lewandowsky), Berlin, 1911, p. 270.

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birth, and gives one the impression of an advanced atrophy or aplasia of muscles; also, the skin over the muscles is usually smooth, peculiarly shiny and colorless.

CASE 2.—E. P. L., seen Sept. 2, 1919, had a very evident right facial paralysis, associated with other interesting phenomena, a description of which I shall give after that of the seventh nerve. I am reporting it here as a congenital condition on the grounds of the convincing statements of the patient, a very intelligent, refined and educated young man. Since his earliest recollections his face has had the peculiarities now present. These he recounted in detail. In careful conversations with his mother she had convinced him and others that the condition was congenital. She had further told him that her labor at his birth was normal in all respects. His mission to me was out of the ordinary. The father of his fiancée had asked him to furnish a certificate from a specialist that his facial condition was not hereditary in character. He had gone about this conscientiously and systematically, by first obtaining all the data he could concerning his family history, and then presenting it and himself to me for examination, coming a long distance in order to do so. This history contained nothing bearing on the case.

The right sided paralysis is evident in all portions of the face, but more so in the lower. In going through the usual test movements the corrugations of the right forehead are only slightly less than on the left side. The palpebral fissure is about equal on the two sides, but there is a retracted and fixed appearance of the right lids in contrast to those of the left. The pulling to the left is marked, causing considerable disfigurement. On the right the lips are slightly flabby, the lower lip being somewhat everted. The whole right side of the face has a thinner and more flabby look as compared with the left side, and this is more marked in the lower portion. The condition causes the patient no disability or inconvenience of any kind that he can mention. The function of the left seventh nerve seems to be wholly intact.

In addition to this right facial paralysis there is evidently some defect in both motor fifth nerves, or at least in some of the muscles which they serve. Both temporal muscles are attenuated, giving a sunken appearance to these regions, and the contractions under the finger are feeble compared with a normal subject. The right masseter, compared with the left, has an hypertrophied appearance. This is probably, partly at least, due to attenuated superstructures. The left masseter is deficient in bulk, especially the anterior portion of it. What there is of it, however, contracts forcibly, as does the right. The lower jaw is slightly shifted to the right, and tilted slightly upward on this side, so that the teeth do not articulate regularly. The line of the lower incisors is one-fourth inch to the right of the upper ones. The lateral movements of the mandible, however, seem normal in range and strength,

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and the same seems to be true of its vertical force, notwithstanding the muscle defects just described. Dr. M. A. Bliss examined the patient with me and called attention to the fact that the platysma function was apparently entirely absent on both sides.

A phenomenon which greatly interested us was a very fine fibrillation, ranging in small waves about the face, almost constantly present in one region or another. This invades more or less all portions of the face below the lower eyelids and seems more pronounced on the right. The masseters appear also to be involved, especially at the acme, as it were, of the larger waves of the movement. The movements are so fine as to be noticeable only when rather close to the patient.

The musculature of all other portions of the body is thoroughly intact. When stripped, he presents a fine symmetrical appearance in this respect. The reflexes in all extremities are normal. He seems well and strong, and is very active. He was through rather intensive service for several months during the war. There is no disturbance in function of any of the ocular muscles, nor of any of the cranial nerves, except those here mentioned.

There are no sensory disturbances anywhere. Taste is not impaired on either side.

In view of these findings it would seem that the defects are nuclear.

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EXPERIENCES WITH LUMINAL IN EPILEPSY

JULIUS GRINKER, M.D.

Associate Professor of Nervous and Mental Diseases, Northwestern University Medical School; Professor of Nervous and Mental Diseases, Chicago Postgraduate Medical School; Attending Neurologist, Wesley, Postgraduate and Mercy Hospitals

CHICAGO

Luminal is comparatively unknown in the treatment of epilepsy. Introduced in this country shortly before the war, its importation was stopped before the medical profession became thoroughly acquainted with its properties. Thus it happened that the claims made for it by German writers in the medical journals of 1912 and 1913 have not been verified until quite recently.

My attention was directed to the value of luminal in epilepsy by the casual reading of an article by A. Hauptmann, published in 1912 in the *Münchener medizinische Wochenschrift,* and confirmed by others, whose bibliography is herewith appended.

Hauptmann selected for his tests cases of epilepsy which had been previously under supervision for several years, or in which an exact history was obtainable. Most of these had received large doses of bromids for long periods. In reviewing his experience he says:

> Luminal tends to exert a favorable influence on epilepsy by reducing the number and severity of the attacks. Its field of applicability will be chiefly in those very severe cases of epilepsy which can no longer be controlled by the largest doses of bromids. In cases of medium intensity the attacks failed to occur under doses of 0.15 to 0.2 gm. (2 to 3 grains) daily, while in the more intense 0.3 gm. (5 grains) need not be exceeded. No injurious by-effects of any kind have been observed after months of daily administration, and especially no cumulative action has developed. Therefore, luminal can advantageously replace the bromids even in the milder cases where these appear contraindicated on account of by-effects.

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My clinical experience with luminal began toward the end of 1913 in the case of a clergyman's son, whose history is as follows:

CASE 1.—A boy, 13 years old, was first seen, June 4, 1913. The family history is negative: Father, a well-balanced clergyman, mother, brothers and sisters are well and there is no epilepsy in the collateral branches of that family. Born in normal labor, patient developed as other children, with diseases usual in childhood, but no serious illnesses. Teething was unaccompanied by convulsions; but there were peculiar spells described by the mother as so-called "inward" convulsion, and certain attacks which were undoubtedly those of "minor" epilepsy. About thirteen months before coming under my observation a severe nocturnal convulsion occurred—the first major attack—and one month before my examination a similar attack occurred. Meanwhile the minor attacks have become quite frequent—five to six daily. In these the patient never falls, but remains standing and appears dazed for a few seconds only. Any slight indisposition such as a cold or indigestion, seems to cause an increase in the number of spells. The examination revealed no sign or symptom of organic disease.

An acne-like eruption covered the patient's face, due largely to the bromids administered for the attacks. My treatment at first consisted in giving the well-known Bechterew mixture—sodium bromid, 15 grains; Fowler's solution, 4 drops, in an infusion of adonis vernalis, three times daily. Though there was improvement, the severe and the light attacks did not entirely disappear, and I decided to try luminal in daily doses of 1½ grains. The effect was marvelous and almost instantaneous. No more attacks were reported and the acne rapidly disappeared. The boy progressed in his school studies. There were no attacks of any kind during the following two years. However, after some indiscretion in diet two slight attacks were noted, but none after that.

Shortly after observing the effects of luminal in this case, I cautiously administered the drug in other cases, with similar results.

In the discussion following Dercum's paper on Epilepsy with Special Reference to Treatment,[1] read before the Section on Nervous and Mental Diseases of the American Medical Association in 1916, I was the first in this country to express an opinion on the use of luminal in epilepsy. Dr. Ball, who participated

1. Dercum, F. X.: Epilepsy with Special Reference to Treatment, J. A. M. A. 67: 247 (July 22) 1916.

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in the same discussion, also emphasized the value of luminal in the treatment of the epileptic attack. At that time my experience was limited to few cases, but since then I had the opportunity of studying over 100 cases of "essential" epilepsy, which constitute the basis for this report.

In order to avoid misunderstandings as to what may be expected from the use of luminal, we must be reminded that in spite of the numerous attempts at a rational, or causal, therapy of epilepsy, most of the methods hitherto proposed have proved fallacious, as has been so well shown by Russell G. MacRoberts. Our therapy is still limited to the prevention of the convulsion or, failing in this, we endeavor to render the attacks less frequent and less severe. The convulsion being only a symptom, though the most conspicuous one, we are far from deluding ourselves into a belief that in causing the arrest of convulsions we have succeeded in curing the disease. But the convulsion, or its equivalent, being nevertheless the one symptom which stigmatizes the individual suffering from it as an epileptic, it is necessary to concentrate our efforts in the direction of ameliorating the epileptic's lot. Though we cannot cure his *disease,* we endeavor to influence the *attacks* which make his life so burdensome.

It would be wasting time were one to enumerate all the drugs which have been recommended for the treatment of epilepsy. It will suffice to state that the bromids have held undisputed sway since their introduction by Laycock in 1857. After a short trial with chloral, borax, nitroglycerin, belladonna, adonis vernalis, and the opium-bromid treatment, the medical profession has accepted bromids as the anticonvulsive remedy of undisputed merit.

Let us briefly examine some of the effects, pleasant or unpleasant, which bromids exercise on the human economy.

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In medium-sized doses, bromids reduce the number of attacks, but they also act as motor depressants and lessen the activity of the intellectual centers. In moderately large doses there is produced apathy, listlessness, lack of interest in one's surroundings, and a peculiar dull expression. After the long-continued administration of the remedy in large doses there occasionally develops a state resembling dementia.

The circulation is often affected by lowering of the blood pressure, cold hands and feet, anemia, and an irritable heart. In toxic doses, arrest of the heart's action in diastole may occur.

The gastro-intestinal functions may become affected by the drug, causing a diminution of the normal secretions of stomach and intestine, thus producing indigestion and constipation.

In those taking bromids in large doses and for a considerable period of time we may note depression, loss of mental and physical strength, forgetfulness, slow speech and retardation of the thought processes. The skin often shows the well-known acne-rash, and even pustules in some cases.

The late Dr. Spratling, certainly an authority on epilepsy, makes the following statement in his well-known book:

> Bromids should be given as a last resort. Cures by the bromid treatment are not to be expected. I know of no drugs, save those which produce habits, such as opium and cocain, that are so universally abused as bromids. The effects of these drugs on the heart, respiration, gastro-intestinal canal, skin and nutrition, are *five* points against their use in epilepsy compared to *one* in their favor, namely, their power of lessening overexcitability of nervous tissue.

While one may not accept in its entirety this author's disparaging remarks on the effects of bromids, it is nevertheless a fact that bromids act unfavorably on many individuals, thus becoming contraindicated in the treatment of epilepsy. Besides, most authors agree that bromids exercise but little influence on the petit mal attacks, and that they are practically useless in the

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psychic forms of the disease and in the so-called psychic equivalents.

Gowers, an ardent advocate of the bromid treatment, regrets that the amount of bromids administered to epileptics at the National Hospital for Paralyzed and Epileptics had to be steadily increased, and that for thirty years the physicians have been constantly endeavoring to discover an efficient substitute for it without success.

There is no doubt of the fact that those having much experience with epilepsy have become disappointed with the bromid treatment and, while still prescribing bromids, are constantly searching for efficient substitutes.

In my opinion a good substitute has been found in luminal, a drug first prepared in 1911 and given to the medical profession in 1912.

Physical Properties of Luminal.—Luminal, whose chemical name is phenobarbital or phenyl-ethyl-barbituric acid, occurs in tablets of 1½ grains and in the form of a white, odorless and slightly bitter powder. It is almost insoluble in cold water, slightly soluble in hot water, soluble in alcohol, ether and chloroform, and in alkaline solution.

There is also a soluble preparation of luminal, *luminal sodium,* which is phenobarbital sodium. This preparation is principally used for hypodermic injection in the form of a 20 per cent. solution, prepared by dissolving the salt in boiled and cooled distilled water. The dose of luminal sodium is 10 per cent. greater than that of luminal.

Actions and Uses.—The drug was first recommended as a hypnotic and substitute for barbital (veronal), whose formula differs from the latter in the fact that one phenyl group displaces one ethyl group, and it is claimed that this change increases the hypnotic power of luminal (phenobarbital) over that of barbital. The dose of luminal as a hypnotic is from 3 to 5 grains, increased if necessary to 12 grains.

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Method of Administration of Luminal in Epilepsy.—I usually prescribe luminal in doses of 1½ grains, either in tablet or capsule, to be given at night before retiring. The patient is instructed to report within a few days. If the dose administered suffices to reduce the attacks or cause them to subside, it is given regularly in the same manner. If this dose is found to be insufficient to cause a cessation of attacks, it must be increased to 2 grains, given at night. Should this dose also prove too small, then another daily dose of luminal is added to the preceding one. In that event my rule is to prescribe a small dose, say 1 grain, to be given in the morning, while the larger dose is administered before retiring. Only rarely have I been compelled to give three daily doses of luminal, but one need not hesitate when it becomes necessary to increase the dose in order to get results. For instance, in one case of severe epilepsy of many years, the patient suffered from six to twelve convulsions daily, and nothing less than 3 grains three times daily caused any change in the patient's condition. After several days or weeks, it will usually be possible to return to the smaller doses, when the effects will be as favorable as with the initial larger doses. When drowsiness and apathy appear, it is always time to reduce the dose. My invariable rule is to give as little as is necessary to keep the patient free from attacks, and no more. In cases that have been treated with bromids continuously for long periods of time it is advisable to begin the luminal treatment with somewhat larger doses at first, to be reduced later. The treatment is continued for years systematically; any interruption is apt to be followed by a number of severe attacks—the same as with the bromid treatment.

The effect of the change from bromid to luminal is especially marked on the mentality of the patient—the manner of speech and general reaction to environmental conditions—who appears to become a different individual.

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A point worthy of mention is the experience that even moderate doses of luminal seem to exert a favorable effect on the minor attacks, while the bromids are comparatively ineffective in these conditions.

Contraindications.—I know of no definite reasons why luminal should not replace the bromids in most cases of epilepsy requiring drug treatment.

Regarding skin eruptions, I can report that among my number of treated cases (100) I have only seen two patients with rashes that necessitated a discontinuance of the luminal treatment.

Urinary disturbances from luminal I have never seen, though continental writers speak of their occurrence and advise as a preventive measure to omit one day's treatment out of the seven.

On the whole, I believe that there are not contraindications to the administration of luminal in kidney or cardiovascular conditions.

To demonstrate more clearly the results of treatment with luminal on the attacks and general condition of epileptics, I have made the following abstracts from the records of several representative case histories.

Case 2.—Miss M. G., aged 23, with negative family and personal history, came under my observation, March 2, 1918. Though her health and development during infancy were of the average kind, peculiar "crying" spells without apparent cause are reported by the mother, and also the circumstance that even slight illnesses were accompanied by physical upset and high temperatures. Menstruation was regular and painless.

At 18, while at college, the first "fainting spell" occurred; the second came one year later. Then followed several similar attacks in periods varying from six months to one year apart. The last attack, one month before consulting me, was attributed by the patient to "shock from dental work."

The first few attacks were accompanied by loss of consciousness, biting of tongue, frothing at mouth and the striking of head against objects encountered in falling. Duration of attack, five minutes; it is usually followed by severe headache and sometimes by sleep. The earlier attacks were more severe than any of the later ones, during the last of which she experienced premonitory warnings in the nature of a "receding" feeling, lasting long enough to enable her to lie down before the attack actually appeared. In addition to the attacks

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described, there are present smaller ones, consisting of peculiar sensations accompanied by dizziness and lasting only a second—evidently "petit mal" attacks.

I prescribed luminal in doses of 1½ grains, to be taken at night. The results were satisfactory. Though the patient has visited me monthly, there has been no record of a single attack since beginning the new treatment. This means that not only have convulsions disappeared, but also the minor attacks.

CASE 3.—I. C., aged 13, schoolgirl, with good family and personal history, was seen, May 6, 1918. Was born in normal labor and developed normally, but suffered from teething convulsions at the age of 1½ years. Six months after the last of these, attacks returned at monthly intervals, then at shorter intervals. The convulsion consists of a short tonic, followed by a longer clonic stage, with foaming at the mouth and the other phenomena of an epileptic attack. There were both diurnal and nocturnal attacks occurring about once in six months when patient was 8 years old. Somewhat later, and up to the time of her first visit to my office, an aura developed before each attack, consisting in a peculiar drowsy feeling. In addition, the serial form of epilepsy made its appearance—there occurred a number of diurnal convulsions following each other rapidly.

Luminal in 1½ grain doses, taken at night, caused a complete cessation of attacks since the first dose was administered.

CASE 4.—W. X., aged 24, with negative family and personal history, was first examined, Sept. 13, 1914. The beginning of his trouble was while a cadet at a military academy; then after a strenuous march he suffered from a so-called "fainting spell." Within a half year five similar attacks appeared in rapid succession. In all of these he became unconscious and keeled over, but consciousness quickly returned. These were undoubtedly minor attacks of epilepsy. His first major attack occurred two years before consulting me; the second one about a year after the first. While the severe convulsions have been coming on an average once yearly, many minor attacks have been noticed in the interval.

The administration of luminal in the usual doses (1½ grains nightly) was followed by happy results: not one attack has appeared in two years, in spite of additional duties imposed on the patient by the necessities of the war. Only once or twice has patient suffered mild attacks while taking luminal—always, however, after disregarding essential hygienic rules.

CASE 5.—O. H., aged 45, came under observation, April 23, 1915. Epileptic attacks appeared since his eighteenth year, which were mild and infrequent at first, but became more frequent and intense later. In the last few years his nocturnal attacks occurred on an average once in six weeks; during these there was complete loss of consciousness, frothing at the

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mouth, and biting of the tongue, with occasional involuntaries. There is no mental impairment, but casual spells of ill temper are mentioned by his spouse. The case was seen by Erb and other European neurologists, who diagnosed "essential" epilepsy.

Owing to the presence of high blood pressure (170 systolic) I though it best to try the combination of bromids and nitroglycerin. Though improvement was marked, attacks continued to come every few weeks. After the systematic administration of luminal in doses of 1¼ grains, mornings and evenings, there have been no attacks in two years and eight months.

Case 6.—J. G., aged 5½ years, was examined, Jan. 27, 1912. First convulsion at 4 years; in the beginning attacks were rather infrequent, but became more frequent later and occurred up to five or six times in twenty-four hours. Though well-developed physically, the boy had always been backward in his school studies.

Bromids were administered in 15 to 20 grain doses three times daily, and long intervals of freedom from attacks were the result. But while still taking massive doses of bromids, the attacks recurred with greater frequency and severity.

January, 1920, I placed the patient on 2-grain doses of luminal daily. There have been no attacks since.

Case 7.—Mrs. J. D., first seen, Feb. 20, 1916, is 54 years old, has been married thirty-five years, and gave birth to three healthy children. The first convulsion occurred only six months before consulting me; three months after the first came a series of convulsions. The examination in this case pointed clearly toward a vascular cause for the epileptiform attacks.

The administration of luminal in 1½ grain doses nightly failed to exert any appreciable effect on the attacks. The dose was consequently increased to 2 grains. There have been no attacks in two years.

Case 8.—A Sister of Charity, 22 years old, was seen June 28, 1918. The first convulsion occurred when she was 4 years old. Following this there was a long period of freedom from attacks until puberty. Then "fainting spells" appeared, and a little later attacks of momentary "abstraction." The last kind of attack occurred very frequently. Still later, regular convulsive attacks came to dominate the scene; at first only once or twice yearly, but recently every seven to ten days.

Luminal in doses of 1½ grains, increased later to 2 grains, nightly, caused a disappearance of attacks during the past one and one-half years.

Case 9.—C. R., aged 5 years, suffered from attacks of epilepsy for six months prior to April 7, 1912, when I saw him for the first time.

Treatment was begun with bromids, and the results were favorable, though there were still occasional attacks. Not

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until I began the administration of luminal did attacks cease entirely for a long period of time. After indiscretions in diet and after illness a convulsion is still recorded, but the average of freedom from one was lengthened from one a month to one yearly.

CASE 10.—H. B., a factory girl, aged 23, under observation since Aug. 27, 1918, had suffered from teething convulsions at 1½ years and subsequently was backward in school. Two and a half years before visiting me she had her first nocturnal epileptic convulsion. There also developed petit mal attacks, consisting of peculiar automatic movements of the fingers, blanching of the face, and momentary losses of consciousness.

After the administration of luminal there were no attacks during a period of three weeks. Patient considered herself cured and omitted taking the medicine for four days. Then a series of attacks occurred, worse than any she had had before.

This case seems to support the statement frequently made, that with cessation of treatment, the attacks recur even in greater severity than before taking luminal.

In this connection it must be stated that several of my patients returned after having taken luminal for a short time believing themselves cured. I always explain to such patients that there is great danger in discontinuing treatment abruptly and that I expect worse attacks than they ever had before, if they omit the remedy. Such a statement often has the desired effect.

In other instances, patients fail to put in appearance and act as though they were cured, neglecting every sort of rule and leading a riotous life. A series of convulsions usually brings them to their senses. Then they visit the physician and promise to be good. For a time rules are adhered to, the medicine is regularly taken, and the patient is again in fine spirits. They often fall from grace more than once, before they finally learn their lesson.

CASE 11.—A housewife, aged 34, first seen, July 13, 1917, gave the following history: After a case of pleurisy she developed peculiar attacks described by her as staring spells, during which the face turns white, then flushes, a jerking of

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hands and feet occurs, and a feeling as though the body was leaving her is experienced. Patient appears to speak to her dead grandmother, wrings her hands continually and asks, "Oh! grandma, what is it?" For all of this the patient herself has no recollection. These minor attacks repeat themselves several times daily. The first epileptic convulsion, a major attack, occurred, Oct. 26, 1916, at night. Subsequent attacks of this type have occurred once monthly and always in the morning. During these she utters a shrill cry, bites her tongue and froths at the mouth; then follows deep sleep. The next day patient is weak and depressed.

The administration of sodium luminal in 2½ grain doses nightly caused a remarkable improvement.

Case 12.—Mr. H. F. B., aged 34, was first seen, Aug. 30, 1918. At 16 years his first convulsion occurred, since which time convulsions appeared at irregular intervals. There are both major and minor attacks. The patient's general health and habits are good, and there is no evidence of syphilis. A relationship has been noticed between his habitual constipation and the frequency of epileptic attacks.

The treatment consisted in regulating the bowels and the administration of luminal tablets in 1½ grain doses. Up to the present time there has been no recurrence of attacks excepting one minor attack. For this reason the dose was increased to 1¾ grains of luminal nightly. There have been no attacks since and the patient is able to attend to the business of a large banking house.

Case 13.—G. M., aged 22, fireman, with good family history, was first seen, Dec. 31, 1919. Suffered from meningitis at 5 years. Shortly thereafter a severe convulsion occurred; similar convulsions returned at irregular intervals, always at night. During these the patient cries, turns his head, stiffens, and bites his tongue. Fatigue and muscular soreness are present for twenty-four hours succeeding the attacks. There were intervals of freedom from spells of varying duration, the longest interval being one year.

Patient was given luminal in 1½ grain doses at night. He reported at my office, Jan. 9, 1920, stating that no attack had occurred since the middle of November. March 10, 1920, the patient reported no attacks since taking the medicine (1½ grains of luminal).

Case 14.—E. L., aged 11, schoolgirl, came under observation, July 9, 1919. Father, mother, one brother and one sister well. The patient was born in normal labor, talked and walked at 14 months; teething at the usual time. As an infant was somewhat undernourished and during childhood suffered from tonsillitis, measles, jaundice, pertussis, scarlet fever, and diphtheria. After a second attack of diphtheria four years ago patient developed a peculiar "stupor." Last autumn suffered from "grip," and her first convulsion occurred in Octo-

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ber, 1918. After passing through a case of influenza, a second attack occurred, December 25 of the same year. Another convulsion occurred, May 30, 1919, and the last attack was observed, July 9, 1919, the day of my examination.

The patient appears active, nervous and tired, and there is seen a more or less constant jerking of facial muscles and of the eyebrows. Mother describes certain peculiar attacks which began four years ago, and which came on while patient was at school. They consist of a momentary confusion when she raises her hand in order to answer teacher's questions. Of late these "absences" have become more frequent. A premonitory symptom noted by mother prior to the development of grand mal attacks is a peculiar stupor when patient first awakens in the morning and a tendency to frequent urination at the same time. Invariably an attack follows the stupor. The attack itself is described as regular tonic, followed by clonic convulsions, lasting from two to three minutes.

Luminal in doses of 1½ grains nightly has caused a diminution of attacks and patient is able to follow her school work regularly. The dose of luminal had to be increased to 1¾ grains before the attacks were entirely controlled.

Case 15.—That in some instances it is necessary to begin with very large doses of luminal in order to prevent attacks and that subsequently smaller doses may suffice to keep the convulsion under control is shown by the case of a feeble-minded epileptic, hemiplegic girl of 10 years of age, who had serial epilepsy consisting of dozens of attacks daily. She was given bromids in large quantities without the slightest benefit. On the administration of ordinary doses of luminal, there was no change. Returning to the bromids, the attacks were as frequent and as violent as before. I then decided to give 3 grain doses three times daily, with the result that attacks ceased at once. After several days I reduced the dose to 2 grains twice daily, and the patient has been free from attacks for an entire year.

Case 16.—That even symptomatic epilepsy responds well to the luminal treatment was demonstrated by the following case: The patient, 52 years old, suffered from generalized attacks of epilepsy during a period of five years, as the sole manifestation of a brain tumor. The administration of even 10 grain doses of bromids caused gastric derangement, extreme weakness, and apathy. I decided to try luminal in doses of 1½ to 2 grains. This and nothing else kept the patient comfortable for many years. In the course of time, however, the patient succumbed from the effects of an inoperable tumor.

Some of my cases have been free from attacks for a period of three to four years, others from one to two years, and still others, and these constitute the

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large majority of cases, for a number of months. Owing to the scarcity of luminal during the war, I was often obliged to use sodium luminal, of which there was still a quantity left. However, the dose is much larger and the effects do not equal those of luminal. Fortunately, both products are now being manufactured in America.

SUMMARY

Luminal in small doses, from 1½ to 2 grains, once or twice daily, has been found capable of causing an arrest of convulsions in epilepsy.

Larger doses than those mentioned are seldom required, but may be safely given.

Large doses may be administered when beginning treatment, especially after sudden withdrawal of the bromid treatment; even in such cases the dose may be gradually reduced to what constitutes an average dose, namely, 1½ to 2 grains.

No harmful effects have thus far been observed from the long-continued administration of luminal.

It does not appear to be a habit-forming drug.

The effect on the mentality of patients taking luminal for epilepsy was surprising. Patients receiving average doses of luminal do not show the peculiar mental torpor of those taking bromids.

25 East Washington Street.

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ABSTRACT OF DISCUSSION

Dr. George A. Moleen, Denver: I can second what Dr. Grinker has so well brought out with reference to the sedative power of luminal in epilepsy, and it seems to be more effective in the severer cases. I cannot agree with Dr. Grinker, however, in that it is free from all danger particularly in the renal cases. I think it participates in the danger common to all the series of barbituric acids, especially in the more commonly used barbital (veronal) as well as luminal and the sodiums salts of these two radicals. I have seen uremia precipitated in a case of renal insufficiency which resulted in death, and I am a bit chary about the use of any barbituric derivative in cases in which this complication is a possibility. On the other hand, it replaces the bromids with less of the mental torpor that is commonly found with them and even is superior to the von Leube modification of the bromid treatment. Even with the addition of the opiates to the bromids I think luminal has a more favorable effect in cases of epilepsy of long standing, but one must be very guarded about its use during convulsive seizures in general for they may possibly be of toxic origin and renal insufficiency may play a part.

Dr. Marvin L. Graves, Galveston, Texas: My own experience with luminal in the treatment of epilepsy is not quite so favorable as that of Dr. Grinker. Unfortunately, we have no definite etiology on the pathologic classification of the epilepsies so that we might know just what we are dealing with and apply our therapeutics accordingly. It

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appears to me that luminal will be found to be definitely an antispasmodic or anticonvulsive possessing a considerable degree of efficacy. A certain number of epileptics that have been under my care have responded to from 3 to 4½ grains administered daily, the convulsive seizures ceasing, the mental symptoms becoming greatly improved and in all respects the patients appearing to be much better. After the patient has taken the drug for a little while, however, and then ceases taking it, it is not long before the convulsive seizures recur. Moreover, I have seen cases in which luminal, administered in doses as high as 7 or 8 grains daily, has produced no cessation in the convulsions, which, as a rule, occur nocturnally, but which are somewhat controlled by the bromids. Luminal is a valuable drug in ½ to 1½ grain doses, but it is not a curative drug in epilepsy; it is simply palliative, and, perhaps, one of the best palliatives.

Dr. Andrew L. Skoog, Kansas City, Mo.: It is the general consensus of opinion on the part of students of this disease that epilepsy should be termed epilepsies rather than epilepsy. There are many different causes for epilepsy; some we are able to discover, and others we are not. These cases are classified as idiopathic or essential epilepsies. My experience with luminal has been favorable in many cases. I had used it for a short time only before the war, then after a certain period I was unable to obtain any of the drug. I did use some luminal sodium, but I did not get the same results with this drug that I had formerly obtained with luminal. I have begun using luminal again, and in one case of a severe type of epilepsy there has been complete cessation of the seizures. From a review of the literature and personal experience with the drug I would say that it is worthy of serious consideration, especially in certain types of epilepsy. However, in view of reports on the toxicity of the drug, we should exercise considerable caution relative to the dosage and period of continuance. I wonder whether Dr. Grinker has had any cases in which he has discontinued the drug for many months, or a year, with no recurrence of attacks. In view of the fact that we favor the term epilepsies rather than epilepsy, the therapeutics of this group of diseases must be multiple. In a certain number of cases surgical treatment is indicated. Hygiene plays an important rôle, particularly as regards diet. I agree with Dr. Grinker that bromids have been used in excess. In Europe it has been the custom to administer considerably larger doses than we use. I prefer sodium bromid. The toxicity of a bromid may be lessened by certain laxatives, particularly cascara and occasionally liquor potassii arsenitis.

Dr. David S. Booth, St. Louis: My experience with luminal has not been as favorable as I had expected; however, the results obtained have probably been as good as those obtained by the use of bromids. It is true that bromids have

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ill effects, but calcium or sodium bromid is the least injurious. In those cases in which the bromids cause pustules, there is usually some focal infection.

Dr. E. Bates Block, Atlanta, Ga.: I have used luminal in more than sixty cases, and my experience has been very favorable. I do not give over 3 grains in each twenty-four hours, and that at bedtime. It does not cure epilepsy, but it has a controlling effect over epileptic convulsions. Luminal has an advantage over barbital and chlorbutanol in that it does not have any effect left over the next day when it is given at night. I have seen no ill effects from luminal. The bromids tend to produce or light up or increase any preexisting inflammatory condition. I would not give bromids in tuberculosis, nor in any inflammatory condition.

Dr. F. B. Wynn, Indianapolis: Generally speaking, we continue the use of any remedy with the idea of diminishing the frequency of epileptic attacks. I do not know of any drug which has been abused more than the bromids. When I was connected with an institution we gave bromids ad infinitum, but the longer I remained, the less I gave, whether for epilepsy or for excited and extreme cases of mental agitation. The mental apathy, the developing transient stuporous state of the individual, impairment of the digestive functions —all came in the wake of the use of bromids. That is what we are apt to do with luminal. It seems to me we are getting away from the fundamental principles in treating epilepsy. We should keep the bowels free by dietetic and therapeutic measures. We overlook the psychogenic factor. Chronic epilepsy patients with dementia go for long periods without any epileptic attacks. Psychology produces that remarkable result. Patients with idiopathic epilepsy, who have been treated by all known measures without getting relief from their attacks, get well following a visit to a religious shrine. It is simply a matter of getting away from an old environment —a different psychology. We should apply the principle in the management of cases of chronic epilepsy. The physician in charge at our institution in Indiana says that there is a great deal in the psychogenic treatment of epilepsy. Vary the patient's occupation and tell him that it will help him, and it will. It is strange what a wonderful effect the change and inspired hope will have upon the course of the epileptic seizures.

Dr. Roy M. Van Wart, New Orleans: About ten months ago I accidentally made the observation that barbital would control epileptiform attacks. Experiments with barbital in small doses produced practically the same results as luminal. I have treated about 200 patients. Patients are usually given from ½ to 2 grains once daily. In most cases it seems to act almost instantly. It reduces the number of attacks very greatly.

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Dr. Julius Grinker, Chicago: Dr. Moleen spoke of convulsive seizures of toxic origin. I do not include these among the so-called epilepsies. I think there is only one kind of epilepsy, and that is, genuine or idiopathic epilepsy. McRoberts wrote a very illuminating article on this subject, which deserves careful perusal. Dr. Graves objects to treatment with luminal because it does not cure. I did not claim to have a cure for epilepsy, but was only offering a substitute for bromid, a drug which has always been used as a palliative. When a patient has no more convulsions it does not mean that he is free from epilepsy. The convulsive phenomenon is only one of several features which go to make up epilepsy. As to whether any of my patients has not been given luminal for a year and remained free from attacks: I have not dared take one off the drug for that length of time because of the fear that convulsions may return. I have had cases in which the omission of treatment for only a short period caused a return of the attack. Dr. Booth stated that his results were not as favorable as mine. His patients were drowsy. It is a question of proper dosage. Again in some isolated cases luminal will not take the place of the bromids. Nobody has claimed that luminal will affect every case favorably. Dr. Booth also spoke of focal infection being present in some cases. So-called epilepsy due to focal infection is not true epilepsy. The bromids do tend to produce acne. Some patients have a susceptibility to drug eruptions, and this we attempt to remedy by the addition of Fowler's solution. With luminal, however, we do not need Fowler's solution. Dr. Wynn is opposed to the continuous use of any drug. He offers objections to the use of bromids, and if these are true, we should discard bromids altogether. If he had any experience with luminal, he would not be able to mention any contraindication to its use, and certainly would be in favor of continuing the drug as long as it makes patients comfortable. As regards cessation of convulsions because of a change to a new treatment, it is true that most of us have occasionally noticed this phenomenon. A patient may say that he has taken bromids for three years but has given them up, and now is free from attacks. Then something happens, and the attacks are worse than ever. There are longer or shorter intervals between attacks not necessarily due to treatment. Taking a patient off the bromids may precipitate an attack of serial epilepsy or he may be entirely free from attacks. This is no argument against the use of luminal, for, as a matter of fact, certain patients cannot take bromids, but do well under luminal.

Barbital is toxic and habit-forming, and in the few cases in which I used it, the convulsions were uninfluenced, whereas luminal has never proved toxic, except in large doses and in a few susceptible individuals.

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ENDOCRINE IMBALANCE IN THE FEEBLEMINDED

OSCAR J. RAEDER, M.D.
BOSTON

The close relationship that exists between the internal secretions and the central nervous system has long been known. The fine changes of temperament associated with the early stages of pregnancy, the varying neurotic conditions, from slight unrest to grave neurasthenia, that result from a dysthyreosis, is common knowledge of the observant internist and the trained neuropsychiatrist. So impressed were the ancients with the relation of the generative and gonadal influences on the mentality that they regarded the seat of hysteria in the uterus and named it accordingly. Cretinism is an example of a recognized endocrinopathy. The constant association of the athyrea with the marked hypophrenia and microsomia, disproportionate tissue growth, and other characteristics, stamp it as a frank ductless gland disease. The marked resemblance of certain factors in other kinds of feeblemindedness with developmental anomalies, uneven growth rate,[1] and various general and special tissue changes to the cretinic hypophrenia, has shown the desirability of further information in regard to the endocrinous factors in feeblemindedness in general.

From the results of careful experimentation in physiology and biochemistry, and accurate clinical observation verified by pathologic study both during life and after death modern scientific opinion is agreed that the development and growth of the animal

1. Goddard, H. H.: Height and Weight in Feebleminded Children in American Institutions; J. Nerv. & Ment. Dis. 39: April, 1912.

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organism is regulated to a great extent by definite and potent highly complex chemical substances called hormones, the internal secretions of the ductless glands. A constant change in the chemical balance occurs with the progressive periods of growth, more or less independent from the daily or continuous physiochemical balancing of the metabolic functions of the cytoplasm constituting the immediate life.

Besides this hormonic or chemical connection or interrelation between the various parts of the body we have another system which binds the parts together, namely, the neuronic or nervous system. It is by a smooth and well-ordered interworking of the neuronic and hormonic connecting systems that a being is kept in normal poise. An interruption of the nervous or electrical control, or a disproportion between the various elementary substances or internal secretions forming the chemical combining system, or an altered condition of one or more of the constituent hormones, will destroy the balance between the two systems and result in an abnormal or pathologic state.

A great deal of attention and research has been devoted to the nervous control both of the body and also its influence on the glands of internal secretion. I now propose to study the relationship to, and effects of, the internal secretions on the nervous system. As regulators of metabolism, to use the words of Noel Paton, they exert an influence on the growth and function of the brain and nervous system in general, as well as on the rest of the organism.

As a basis for this study, 100 cases of feebleminded-ness that came to necropsy have been taken in chronological order from the pathologic service of the Massachusetts Commission on Mental Diseases. They include cases from the Massachusetts schools for the feebleminded at Waverley and Wrentham, and from other state hospitals.

The cases had been studied clinically in the routine method at the various institutions and after the stand-

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ardized system developed by Dr. W. E. Fernald at the Waverley School, including Binet-Simon intelligence ratings. The necropsies were complete, including head, cord and soma in all cases, and were performed by Drs. M. M. Canavan and D. A. Thom according to the inclusive method outlined by Southard. The data, clinical and postmortem, were all collected in the usual way, symptoms and signs and pathologic changes and findings being noted by the regular examination methods. Thus, neither during life nor at necropsy was there any particular or special attention given from the endocrine point of view.

Data have been collected from a gross pathologic study of this series of brains and somatic organs, especially the ductless glands. In one third of the cases a microscopic examination of the glands was also made. Aplasia, hyperplasia, pigmentation and interstitial change not due to age, glandular proliferation, anomalies such as absence or accessory organs with lessened or increased function, besides special changes in the secreting epitheliums and cells of the individual and various glands are the main factors included in our summation of the changes.

In a previous study,[2] the question of syphilis was considered. Attributing inflammatory and exudative changes, such as early arteriosclerosis, aneurysm, chronic periostitis, saddle nose, chronic meningitis and vascular changes of a doubtful nature to syphilis, we found eleven cases among those in which there were gland changes. Wassermann reactions were not obtained in many of these cases, so that reliance on grosser pathology was necessary.

The incidence of glandular change is shown in Table 1. Under the designation "extreme changes" are classed those cases in which three or four glands were involved and in which there were marked anom-

2. Raeder, O. J.: A General Nosological Grouping of One Hundred Autopsied Cases of Feeblemindedness, presented before the New England Society of Psychiatry, September, 1919.

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alies of growth, underdevelopment, disproportion of body parts, such conditions as dystrophia adiposo-genitalis, etc. There were two cretins.

TABLE 1.—GLAND CHANGES

	Number
Extreme	10
Marked	11
Moderate	53
Absent	26

In the second group eleven cases showed marked endocrine signs, in which at least two glands were involved, correlated with distinct changes in growth and anomalous development. Combining the first two groups, we have 21 per cent. of cases showing presumptive evidence of marked internal secretory disturbance.

The third group included fifty-three cases in which changes of one or two glands were demonstrable, with irregular and unusual combinations.

Gland changes were entirely absent in twenty-six cases.

TABLE 2.—PERCENTILE ANOMALIES

	External	Visceral	Cerebral
Paresis (100 cases)	23	29	27
Hypophrenia (60 Cases)	73	58	75

A tabulation of anomalies (Table 2) divided into external somatic, visceral, including the ductless glands, and cerebral, showed an interesting comparison. In the absence of data on normal individuals, 100 cases of paresis were selected. Paresis being in each case an acquired disease, it was assumed that they might be potentially representative of the normal type.[3]

The marked preponderance of anomalous manifestation in the feebleminded over the cases of acquired disease is striking. Attention is called especially to

3. Only sixty of the hundred cases were selected to compare with the 100 cases of acquired disease because the combined 160 necropsies were performed by the same hand, insuring a more even and regular observation.

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the third or cerebral group, in which 75 per cent. of hypophrenics showed anomalies, compared with only 27 per cent. of the acquired-disease cases, as significant of a possible influence of the growth regulating secretions on the development of the brain.

Among anomalies of development the stature first demands attention. Our observations on height agree in a general way with those of Shuttleworth,[4] Tarbell,[5] Wylie[6] and the compilation of statistics by Goddard[1] in that we found the majority of patients undersized. In Table 3, percentile figures show 60 per cent. varying from the normal, of which 51 per cent. were undersized and 9 per cent. taller than the normal given for their ages. Thirty-eight per cent. were normal, allowing a variation of 10 cm. as within normal limits. Data were not available in 2 per cent.

TABLE 3.—HYPOPHRENIA: STATURE IN ONE HUNDRED CASES

	Per Cent.
Undersized	51
Oversized	9
Normal (± 5 cm.)	38
Unknown	2

The question of growth has been studied experimentally in tadpoles by the feeding of various ductless gland substances in the laboratories of Gudernatsch and others. Gudernatsch[7] has shown that tadpoles fed with thymus are overgrown and underdeveloped; also that examples of the same hatching fed on thyroid develop more quickly, but at the expense of size and weight.

4. Shuttleworth, G. E.: Health and Development of Idiots Compared with Mentally Sound Children, Proc. Assn. Med. Off. Am. Insts. Id. & F. M., 1876-1886, pp. 315-322.

5. Tarbell, G. G.: On Height and Weight and Relative Rate of Growth in Normal and Feebleminded Persons, Proc. Assn. Med. Off. Am. Instns. Id. & F. M., 1876-1886, pp. 188-189.

6. Wylie, A. R. T.: Contribution to the Study of the Growth of the Feebleminded in Height and Weight, J. Psyc.-Asthen. 8: 2-7 (Sept.) 1903.

7. Gudernatsch, J. F.: Feeding Experiments on Tadpoles, Arch f. Entwickl. d. Organ, 35, No. 3 (Dec.) 1912; Am. J. Anat. 15, No. 4 (Jan.) 1914.

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In the curves on growth, Tarbell,[5] and especially Goddard,[1] have shown that hypophrenics grow faster but stop sooner than normal children. Allowing for differences in comparative anatomy, the question arises whether this phenomenon may not be due to an imbalance of the thymus and possibly later also of the pituitary gland. Certainly the same overgrowth of the thymus-fed tadpole corresponding to increased height and weight in the preadolescent stage of the feebleminded, as shown by Goddard's tables, and the underdevelopment or immaturity of the tadpole can be correlated later with the stunted development and hypophrenia of the abnormal child.

TABLE 4.—ADENOSES

	Per Cent.
Thyroid	19
Pituitary	40
Suprarenal	27
Sex	38
Thymus	12
Others	6

The incidence of changes in the individual glands is shown in Table 4. The pituitary gland was found involved oftener than any other gland, in 40 per cent. of cases. The sex glands, the seat of numerous anomalies which brought their score up considerably, were second with 38 per cent. The suprarenal showed involvement in 27 per cent. of the cases and the thyroid in 19 per cent. It is noteworthy that the thyroid falls so low in the scale. With the frequency of thyroid disturbances one might expect a larger number of cases showing a pathologic condition of the thyroid.

Numerous cases showed various combinations of glands involved. Table 5 gives the pituitary polyadenoses. It was involved singly in fourteen cases. Pituitary with gonads, in nine cases, was the most common dual adenosis, though there were combinations of sex and thyroid in four instances, sex and suprarenal in four cases, and in three cases the thyroid

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pituitary and gonads were affected in triple involvement.

Furthermore, there were six cases in which the gonads were combined with three other glands; two included the gonads, thyroid, pituitary and suprarenal; two, gonads, thyroid, pituitary and thymus.

TABLE 5.—POLYADENOSES

	Number
Pituitary	14
Pituitary, Sex	9
Pituitary, Suprarenal	5
Pituitary, Other Glands	11

Of the third two in which thyroid, pituitary, suprarenal and sex glands were involved, one was a female cretin, and the other an obese eunuchoid idiot who had been castrated in early life. He showed all the typical characteristics — undeveloped penis, and feminine appearance of breasts and extremities, with flabby pendulous abdomen.

REPORT OF CASES

In order to illustrate the striking endocrine characteristics that were found in many cases of idiocy and imbecility, two of the cases have been individually charted and are shown in Figures 1 and 2.

CASE 1.—*History.*—Figure 1 represents a male idiot, aged 10 years. He was admitted to the Massachusetts School at the age of 6. He was 3 feet 9 inches tall and weighed 43 pounds; he was poorly developed and nourished, had chronic bronchitis, had a dry, coarse skin and sparse, coarse, wiry hair; there was a systolic heart murmur at the apex; the genitalia were infantile; there was an umbilical hernia, a protruding abdomen and poorly developed muscles; the hands were thick and short, with stubby fingers; the forehead was bulgy, the face asymmetrical, with large protruding ears; there was a chronic discharge from the nose; the tonsils were enlarged, with adenoids and a fissured tongue. There was some question whether or not the patient was a cretinoid idiot.

There were six children, of whom the patient was the second. The first three children resembled one another rather closely, and all died at the Massachusetts School for the Feebleminded. Three later children were normal, one dying

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in infancy of diphtheria. An uncle and a sister of the patient had spinal trouble; the sister in question became partially blind. There was a feebleminded and epileptic great-aunt. One of the mother's sisters was migrainous and obese; one living child of the sister had one arm not fully developed. Both the patient's mother and maternal grandmother are stated to have been nervous, and the patient's grandmother was also migrainous. On the father's side one sibling was deformed with spinal curvature. There was also an epileptic and feebleminded great-aunt.

The patient began to talk at about 1 year and to walk alone at 19 months. Peculiarities were first noted at 2 years. The patient had a peculiarity in speech, using "d" for the first letter. He was able to help in dressing and undressing himself and to feed himself with a spoon or fork.

School Progress, School Examination, Practical Knowledge and *Economic Efficiency* were all nil.

Social Reactions.—The patient was stubborn and irritable, running about in his own way, screaming when touched to be dressed or undressed, spitting on the floor, and yelling if he did not wish to be moved; he would not eat if any one was looking at him; he had lost capacity to feed himself, throwing food about; he was attracted by striking colors in dress. If given a toy, he would go by himself to play with it, screaming if another child went near him.

Psychologic Test.—The Binet test, performed in May, 1914, at the chronological age of 8 years, gave the mental rating at 1 year.

Further History.—The patient required nursery care. He was in the hospital and infirmary most of the time, with untidy habits. He had frequent illnesses, including chicken-pox, measles, bronchitis and indigestion; he grew thin and developed a cough in November, 1915; he had a slight unilateral convulsion with twitching of the right side of the face. There was severe dyspnea with mucus in the throat and a weak pulse. The patient showed symptoms of meningitis and remained comatose. Three days later slight paralysis of the right side developed, followed by death the next day. The cause of death was meningo-encephalitis and hydrocephalus.

In Figure 1 are shown, first, the small stature and general microsomia; secondly, hydrocephalus; this was considered as being evidence of syphilis. A postmortem Wassermann test was negative on the serum; thirdly, various dystrichoses, namely, hairy growth over the chest and abdomen, upper arms and forearms; abundant hair on back and neck, with absence of crines pubis and axillary hair. Over the head it was coarse and rough and arranged in multiple cowlicks; fourthly, microphallus, the penis being small with redundant prepuce. Besides these anomalies and abnormalities, the

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pituitary was cystic, and there was little sign of activity in the secreting cells of the anterior lobe and marked increase in the pigmentation of the posterior lobe cells.

CASE 2.—*History.*—Figure 2 represents a female Mongolian, aged 18 years, who rated 3½ by the Binet intelligence scale. She was admitted to the Massachusetts School at 8 years of age. At 17 she was 4 feet 6½ inches tall and weighed 72½ pounds. She later increased 12 pounds in weight. The patient was fairly well developed and nourished; she was a mouth breather. The skin was coarse, the circulation poor; the heart sounds weak, with systolic murmur at the apex; the heart was enlarged; the fingers and toes were short and blunt; the face was asymmetrical; the eyelids were inflamed, the nose flattened, the tonsils large, and the tongue fissured. The Wassermann test was negative.

ENDOCRINOSIS
IDIOT

MICROSOMIA
HYDROCEPHALUS
DYSTRICHOSES
MICROPHALLUS
HYPOPHYSITIS
LYMPHNODITIS

Fig. 1.—Case 1.

ENDOCRINOSIS
MONGOLIAN IDIOT

MICROSOMIA
HYPOTHYREOSIS
HYPO-ADRENIA
HYPERTHYMIA
OVARITIS
HYPOPHYSITIS
LYMPHNODITIS
DYSGENITALISM

Fig. 2.—Case 2.

The patient was the first born of two children. The younger brother is normal. The father had an enlarged heart; he was of a gouty, asthmatic diathesis and died of a paralytic shock. He had an epileptic seizure at the age of 30, and two more after marriage, one just previous to the birth of the patient. He had a violent temper and was subject to severe headaches. The parental fraternity was further checkered with nervosity and paralysis agitans, and there was a question of insanity. On the mother's side two brothers were epileptic, and a sister was backward. Two brothers and one sister are described as normal. A distant cousin had schizophrenia, and her brother was hypophrenic.

Peculiarity in the patient was noticed at 1½ years. She began to walk and talk at 2½ years; she was never able to say such words as "or" and "if"; she did not know the alphabet and could not read or count. She was tidy and could aid in dressing and undressing herself; she could use a spoon but not a knife and fork, nor could she tie her shoestrings. She was described as sometimes obedient and as being fond of picture books and blocks.

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School Progress and *School Examination* were nil.

Social Reactions—The patient was quiet and inattentive; her name had to be spoken to attract her attention.

Waverley School History.—The training class records show a deficiency in industrial adaptability. At 12 years she was described as clever at playing simple games, catching a ball, etc. Later on, after the age of 12, she learned to sew and use scissors well. From 1905 to 1909 (age 9 to 13 years) she showed slow but definite improvement. She had changed under training from a repulsive, drooling, finger-sucking, untidy, stupid patient, taking short, choppy steps, to a tidy patient who had completed the first school course and had begun to take part in manual training. Her resistance to disease was low; she was subject to infections of various kinds. She was admitted to the hospital ward, April 23, 1915, with severe bronchitis and pleurisy. She died of tuberculous bronchopneumonia, May 26, 1915.

She was of the usual Mongolian type with the characteristic tissue and developmental changes. In Figure 2 we have abridged the enumeration of these characteristics, showing microsomia, very small thyroid and suprarenals, hyperplastic thymus, an interstitial inflammation of the ovaries and hypophysis, a general lymphnoditis, including tonsils and adenoids and dysgenitalism in the form of unequal labia majora, long urogenital cleft, large, prominent clitoris and a high position of the mammae.

Three of the Mongolians showed extreme glandular change; one showed marked involvement, and the fifth one moderate dysadenosis. Thus all five cases of Mongolianism showed gland changes besides the usual typical characteristics.

SUMMARY

In this study, the developmental, tissue and gland changes have been charted in 100 cases of feeblemindedness that came to necropsy, taken in chronological order from the pathologic service of the Department of Mental Diseases of Massachusetts. Data found in the case histories and necropsy protocols were gathered in the regular way without reference to the endocrine point of view. It has been shown that 21 per cent. of cases showed marked glandular involvement, with changes of a lesser degree in 53 per cent. additional, making a total of 74 per cent. in which there were signs of gland change. In eleven

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of these there were signs of syphilis. Not having Wassermann reactions in all cases, it may be argued that others of the glandular cases may have been syphilitic. Vice versa, some of those designated as syphilitic because of vascular or chronic inflammatory or exudative changes, may not have been syphilitic. Furthermore, the dysfunction of the endocrine system itself may be due in these cases to syphilitic influence on the secreting cells of the glands. In Figure 1, though the hydrocephalus points to a possible syphilitic involvement, the Wassermann reaction was negative.

The pituitary has been found invaded more often than any other gland. The small stature so common in the feebleminded may be correlated with this fact, in view of the powerful influence of the pituitary gland on growth.[7]

The gonads were shown to be affected in 38 per cent. of cases. Also anomalous development was most rampant here. This is not remarkable when the embryology of the genital tract is considered. The similar, not to say identical, development of the urogenital tract in both sexes up to a certain embryologic stage, and then the more or less arbitrary determination for male or female would seem to give much more opportunity for anomalies, misdevelopments and malformations than in other parts of the organism where the developmental homonomy is not interrupted.

Macroscopic studies of the brain anatomy have shown numerous reversive types and evidences of arrested development or maldevelopment. Microcephaly, especially in the Mongolians, was common. Macrogyria, in one instance almost to absence of any fissuring in the frontal regions of the two hemispheres, was of frequent occurrence. Extraneous fissuring, "affenspalte" lateral inequality, microcerebellum correlated with sprawling gait, and deficiency of parietal and occipital lobes with various degrees of exposure of the cerebellum, were some of the more striking changes found in the encephalon.

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MICROSCOPIC EXAMINATION

Among the microscopic findings, interstitial change, aplasia, pigmentation and varied staining reactions were noted. The pituitary was frequently found cystic; especially dilatation of the cleft in the pars intermedia. The cells of the anterior lobe showed varied staining reaction, the most common change being a deficiency of basophil cells. In the posterior lobes, marked pigmentation occurred in several instances in very young individuals.

The aplastic condition of the thyroid was present in the cretins. A hyperplasia of the glandular epithelium was noted in several cases, and increased interstitial elements were also common. In one of the Mongolians an infantile condition of the thyroid with absence of colloid was noted. In a few cases the vesicles were distended with colloid.

The tests usually showed signs of activity in all except the lower grades. The idiots' testes were either inactive and fibrous, or only feeble signs of spermatogenesis were found. The ovary was examined in only a few cases, but seemed less virile than the male gonads. Two cases of imbecility showed no ovarian activity, and in a third case only very slight evidence of function, one graafian follicle in a section, was found.

No characteristic changes were found in the suprarenal gland. In one case—a tuberculous subject—there was considerable involvement of both suprarenals with tubercles, numerous giant cells, etc. There were no signs of Addison's disease present.

CONCLUSIONS

1. The evidence of gland changes observed in these cases by routine examination methods, clinical and postmortem, without particular reference to endocrinology, is so constant and multifarious that we cannot but regard them most seriously. There were gland

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changes of one sort or another in 74 per cent. of cases. Marked gland changes occurred in 21 per cent.

2. With the constant and characteristic bony and soft tissue changes microsomia, lowered resistance to infection, poor circulation, loose jointedness, and changes in the glands of internal secretion, Mongolian idiocy bids fair to be founded on an endocrine pathology.

3. The internal secretions begin to exert their influence early in the life of the organism. It is known that permanent adjustments of the other glands and tissues follow on the absence or dysfunction of one gland or set of glands. In order to avoid such permanent changes as infantilism, dwarfism, acromegaly, microcephaly and feeblemindedness, it is imperative that these conditions be recognized and remedied by supplying the deficient hormone or inhibiting the hyperfunction of a gland early in the course of the disease. After permanent adjustments have formed, improvement is difficult; with early treatment, results are often little short of marvelous.

4. Much of the finer pathology of the ductless glands is concerned with biochemical reactions. Further studies of feeblemindedness by physiochemical and roentgenologic research would no doubt throw more light on this obscure field.

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ABSTRACT OF DISCUSSION

Dr. Tom A. Williams, Washington, D. C.: In the attempt to establish a correlation between endocrine deficiency and poor cerebral development we must avoid the danger of the *post hoc, propter hoc* argument. Dr. Raeder's theory may be tenable, but two objections occur to me.

First, in relation to syphilis. Syphilis affects all structures of the body and the feeblemindedness in congenital syphilitics may be primarily cerebral. Therefore, in estimating a statistic it is perhaps fair to eliminate the cases of syphilis. Secondly, we should have a control, namely: postmortem examination of the bodies of a large number of dead persons. Why say that in 100 necropsies certain evidences were found which tend to establish a correlation between endocrine deficiency and feeblemindedness, when these findings may be the effect of the disease which has terminated in death? These cases must be eliminated. In regard to the microsomia of these patients, is it necessary that it be attributed to hypoendocrinia? Perhaps not. Anomalies of growth are the result of endocrine disturbances; but is the growth of the individual who grows without anomalies dependent entirely on endocrine factors? It is doubtful whether we can include microsomia as one of the effects of endocrine disturbances.

Dr. Gustave Roussy, Paris, France: (Translation by Dr. Hitchcock): We must be cautious in our conclusions regarding the rôle of the endocrine glands, not too hastily deciding as to the absolute relations of cause and effect in regard to polyglandular syndromes. Indeed, if one examines systematically the endocrine glands at necropsy on subjects dead of the most diverse affections, as I have done in more than 200 cases, there will be found lesions of several internal glands not usually occurring in this type of patient. Besides the endocrines proper, other tissues are concerned. In a case of Parkinson's disease I found lesions of the parathyroids. The causes of lesions of the endocrine glands, as shown at necropsy, are numerous, complex and inconstant, therefore it is prudent not to be hasty or final in drawing conclusions concerning these lesions.

Dr. Walter Timme, New York: The main difficulty with regard to all internal glandular disturbances is that we do not have a definite static series of organs. The endocrine glands are all of a character designated in mathematics as variables; there are no constants. In order to solve an equation or series of equations which involves variables you must have one more equation than you have variables. Here we have from ten to fifteen variables and only one equation. Hence, we cannot solve the problem. We can only approximate to a solution. If Professor Roussy has examined at necropsy 300 cases of various diseases in which the endocrine glands were found to be involved, then these disturbances

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are part of the process of the disease. They may have been produced by the disease and they may have indicated the lethal termination. In other words, they are terminal states. But when Professor Roussy states that the presence of such lesions in the feebleminded is nonconclusive as to the pathogenesis of feeblemindedness, I can only ask, Do they not suggest a possible cause? Their absence would beyond peradventure be regarded as conclusive! Dr. Raeder claims that in 100 cases of necropsy on the feebleminded he has seen a large proportion that showed definite changes in the internal glandular system. He has approached the subject in a different way than it has ever been approached before, and he has arrived at a definite, high percentage of disturbance in the endocrine glands. His work presents a distinct advance, a new angle of attack in this problem that hitherto has been attacked entirely on the psychogenic side with practically little result. If these disturbances of the internal secretory organs produce idiocy, why does not every disturbance of the internal glands produce idiocy? For this reason: It depends on how the glands are constellated. There are certain constellations of these glands which will produce a condition of diabetes, another which will produce giantism, a third acromegaly, a fourth Addison's disease, etc. It depends on the subject's reaction to these disturbances as to which disease will develop. I tried to bring this out in a table of endocrinopathic inheritance some years ago. I examined every member of a family through four generations. The original parents were a giant and a diabetic. Through four generations there were all kinds of endocrine disturbances varying from exophthalmic goiter to microsomia, and including two morons. Heretofore in investigating ancestry all we looked for was mental disease. That is wrong; mental disease is only one phase of a disturbance, just as is epilepsy.

Dr. M. A. Bliss, St. Louis: It is only by such extensive studies as this, made under opportunities that come only to those who work in institutions of that size, that we shall be able eventually to arrive at some conclusions in regard to these things. Dr. Raeder has made a definite advance toward, perhaps, finally reaching some conclusions about the association of the endocrine glands with these mental deficiencies and disturbances.

Dr. Andrew L. Skoog, Kansas City, Mo.: The brain specimens presented by Dr. Raeder are suggestive of hereditary disturbance or secondary anomaly of the brain developing at some early stage. Those who have studied the cases of various types of idiocy will agree that the patients reach full development at an earlier age than does the average individual, and probably more so as related to the central nervous system.

Dr. Karl A. Menninger, Topeka, Kan.: Work like this furthers the study of the interrelationships of physical and

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mental disease. We have too long regarded the patient as carrying his brains in one basket and his visceral organs in another, and from our standpoint we have looked only at one basket. Endocrinologists deserve praise for calling attention to their field of the human organism. We have ignored a relationship which is far closer than we know at present, far more intimate and intricate, perhaps, than we even suspect.

Dr. Oscar J. Raeder, Boston: Syphilis is to be considered in all cases of feeblemindedness. Fifteen per cent. of our 100 cases were syphilitic; only two of the twenty-one endocrine cases were suspiciously syphilitic, but we had no Wassermann in these cases. Some of these cases date back as far as eight or nine years, before the Wassermann test was so regularly done as now. In necropsies on the insane and the feebleminded we frequently find gland changes and many of them are terminal changes. I would not assign that as a cause of feeblemindedness if the patient had been feebleminded. Such lesions frequently are found in necropsies, especially in senile cases. Dr Timme regards feeblemindedness as one manifestation only in an endocrinologic heredity. In every biologic study the relation of the endocrinologic factors to growth and development is very important, as has been shown in experimental work on the frog. The frog fed with thymus gland was overgrown but underdeveloped, while the frog fed with thyroid gland was overdeveloped but undersized. That point was brought out by Dr. Timme in his example of four generations of one family in which there were two morons, some acromegalics, some giants, while some showed other gland changes. I make no claim that endocrinologic factors are the cause of all feeblemindedness. I simply call attention to the marked gland changes found in all cases of feeblemindedness. I would be glad to find one definite glandular syndrome as the cause of one definite feeblemindedness. There are many feeblemindednesses, such as syphilitic, glandular, traumatic, etc. Dr. Menninger and Dr. Skoog emphasized the fact that in the past the nervous system has been studied separately from the rest of the body. We have not sufficiently considered the body as related to the central nervous system. As Dr. Southard used to say, we look at the neurons, but the body is connected up not only by neurons, but also by hormones, and these must be considered together.

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SYMPTOMATOLOGY OF SPINAL CORD TUMORS

ISADOR ABRAHAMSON, M.D.
Attending Neurologist, Montefiore Home; Associate Neurologist, Mount Sinai Hospital

AND

HYMAN CLIMENKO, M.D.
Adjunct Neurologist, Mount Sinai Hospital and Montefiore Home
NEW YORK

Since 1888, when Sir Victor Horsley showed that spinal cord tumors can be successfully removed, neurologists the world over have attempted to establish criteria by which an early diagnosis may be made. Classical works on the subject have been written, and still the fact remains that spinal cord tumors are frequently overlooked and often not found till late in the course of the disease or only postmortem. It has been our motto to regard with suspicion any transverse myelitis of progressive character. In these cases very careful anamnesis and painstaking examinations have done much to solve some of the problems. We have therefore elaborated the following rather terse and compact scheme which we trust may be of as much assistance to others as it has been to us. Our percentage of correct diagnosis and correct localization has risen considerably. Tumors of the spinal cord, in brief, are extradural, extramedullary and intramedullary. In this communication, we will deal only with the latter two varieties.

By far the most frequent and the most important, from a diagnostic and therapeutic point of view, are the intradural and extramedullary tumors. The symptomatology of this class of tumors divides itself into two main groups: (1) early, or root symptoms, and

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(2) cord compression, or later symptoms. A third group of symptoms may be mentioned, namely, those signs referable to bony, vertebral involvement.

In extramedullary tumors, we differentiate signs of tumors whose density is greater than that of the cord from those whose density is equal to or less than that of the cord.

This distinction has not hitherto been sufficiently accentuated. To our mind, this difference is of vital importance in recognition of tumors of the cord. It is especially true of cases of the softer tumors, in which most of the errors of diagnosis and localization have been made.

Extramedullary tumors whose density is greater than that of the cord show, in brief, these signs:

1. Root signs:
   (*a*) Muscular spasms and twitchings.
   (*b*) Pains.
   (*c*) Paresthesia.
   (*d*) Hyperesthesia.
   (*e*) Changes of reflex, both deep and superficial, at the seat of the tumor.

2. Signs of moderate compression:
   (*a*) Homolateral paresis below the seat of the lesion.
   (*b*) Homolateral hyperflexia and hypertonia below the lesion.
   (*c*) Alterations of superficial reflexes of the homolateral side below the lesion.

3. As the compression increases, further and marked evidences of tract interferences:
   (*a*) Paralysis of the same side.
   (*b*) Marked spasticity, with exaggeration of tendon reflexes and clonus; Babinski and other pathologic plantar reflexes on the same side.
   (*c*) Beginning hyperreflexia and hypertonia of the opposite side below the level of the lesion.
   (*d*) Beginning disturbances of pain and temperature sense on the heterolateral side, beginning at the lowest segment and slowly climbing upward.
   (*e*) Impairment of the vibratory, postural and localization sense homolaterally.
   (*f*) Difficult, delayed and interrupted micturition and constipation.

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4. With still greater cord compression:
   (*a*) Paralysis of both sides below the level of the lesion and more marked at the homolateral site.
   (*b*) Tract sensory disturbances (pain and temperature) on both sides, but more marked on heterolateral side and reaching its maximum over the sacral and lower lumbar distribution.
   (*c*) Retention of urine and feces.
   (*d*) Bilateral loss of superficial reflexes, bilateral, Babinski, etc.

5. With very marked cord compression, signs of a transverse myelitis:
   (*a*) Pain and temperature disturbances now at their highest level, corresponding physiologically to the seat of the lesion (from two to three segments below the actual seat).
   (*b*) Complete bilateral spastic paralysis.
   (*c*) Complete retention of urine and feces, with overflow incontinence.
   (*d*) Beginning disturbance of the tactile sense.

6. Lastly, signs of complete transection of the cord:
   Complete bilateral flaccid paralysis with abolition of all the deep and superficial reflexes; incontinence of urine and feces; complete anesthesia, analgesia and thermesthesia; marked trophic changes.

The level remains practically unchanged throughout the course of the disease.

A tumor with density equal to or less than that of the cord causes little interference of the cord by pressure.

Early anterior or posterior root signs with a belt of hyperesthesia are rarely as marked as in hard tumors. This hyperesthesia is frequently transient and may be easily overlooked. The root symptoms frequently appear earliest over the distal distribution of the nerve.

Cord signs are due to interference by the tumor mass with the lymphatic and venous circulation of the cord and result in secondary edema. A soft tumor can reach a considerable size and slowly mold itself around the cord without causing any marked signs of tract involvement.

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All the motor tract signs enumerated in the hard cord tumors are also present here, only to a milder degree. Both the tract motor and sensory signs, though less in degree, are most marked at the site or just above and below the lesion, and spread downward with diminishing intensity. In hard tumors, it will be remembered, the tract sensory disturbances are greatest in the lowest spinal segments. In soft tumors the tract disturbances are just above, at and below the seat of the tumor. The spread of signs and symptoms is very suggestive of intramedullary growths and syringomyelia.

In the soft tumor group, there is a considerable parallelism between the motor and sensory signs in contrast to the more marked and earlier motor tract involvement seen in cases with hard tumors.

In contradistinction to hard tumors, the level symptoms are not stationary. The upper edge of the level may move for about two or three segments higher.

The presence of xanthochromia in the cerebrospinal fluid is more common in soft tumors.

It is in soft tumors, especially with few signs of cord compression, that a lumbar puncture is likely to be followed by the first absolute evidences pointing to a level of cord compression. Another peculiarity is that in the soft tumor, the root signs are not infrequently bilateral as in contrast to hard tumors, in which until late in the disease they remain unilateral. There is also found in the soft tumors, before operations, remarkable remissions and exacerbations in the course of the disease. A resemblance to multiple sclerosis and syphilis must be borne in mind.

DIFFERENTIAL DIAGNOSIS BETWEEN EXTRAMEDULLARY SOFT TUMORS AND INTRAMEDULLARY TUMORS

1. Pain is present from the beginning in all soft tumors, but only in those intramedullary tumors in which the tumor reaches the posterior roots or surface

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of the cord. Pain in intramedullary tumors is, therefore, usually a late phenomenon, or may not occur at all if posterior roots or the meninges are not affected.

2. In the vast majority of cases, the symptoms develop more rapidly in extramedullary soft tumors.

3. Tract sensory signs and symptoms are more intense and more widespread in intramedullary tumors.

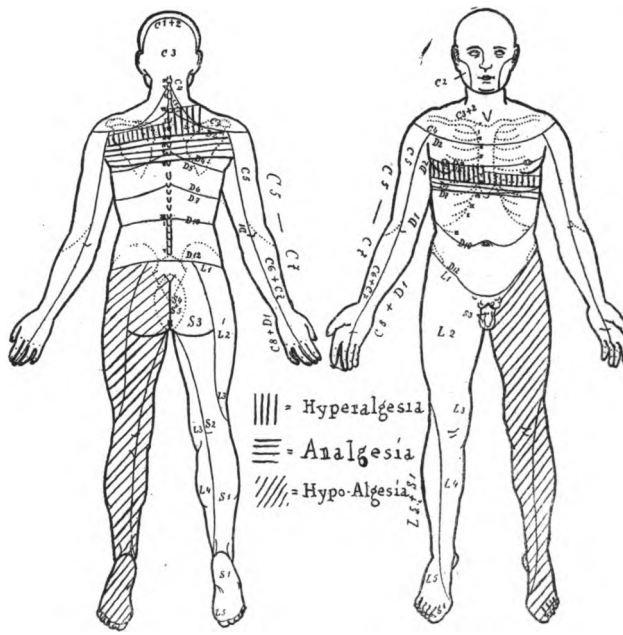

Chart 1.—Sensory disturbances in Case 1.

4. Trophic changes are greater in intramedullary tumors.

5. Intramedullary tumors show less rectal and vesical signs.

6. Xanthochromia is rare in intramedullary tumors. It appears only when the tumor reaches the surface of the cord.

7. Deep spinal column tenderness indicates rather an extramedullary tumor.

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REPORT OF CASES

In the brief space at our disposal, it was impossible to deal with all the details of our histories of cases of spinal cord tumors. It has been our object, therefore, to condense the records as far as possible, the histories of all our cases, giving only their salient features, their chronological succession of symptoms and signs. Other cases and further studies have been reserved for a later publication.

CASE 1.—H. R., man, aged 38, widower, salesman, admitted, Nov. 24, 1913, discharged, July 1, 1914 who presented a negative family history, and who had had gonorrhea several times, had noticed a tickling sensation over the right leg, three years prior to entrance and two months later, "soreness" over the right leg. At about the same time he noticed that while taking a bath the upper part of the body was more sensitive to heat than the lower. Four months later, his right knee became stiff and he experienced twitchings in the right leg. Two months later, the left knee became stiff. Still later, urination became frequent and there was difficulty in locomotion.

At this time the patient was admitted into a hospital, where he was carefully examined. The findings there in brief were: absence of both lower abdominal and cremasteric reflexes; a spastic paraplegia with signs more marked on the right than on the left side; a broad belt of hyperesthesia between the seventh and the tenth dorsal segments, and a line hypalgesia down to the twelfth dorsal segment. Spinal cord tumor was diagnosed. The operation performed extended from the tenth to the twelfth dorsal lamina. No tumor was found at the site.

Nov. 24, 1913, the patient was admitted to the Montefiore Home with a diagnosis of syringomyelia. Here painstaking and careful examination revealed a level of hyperalgesia corresponding to the fourth dorsal segment on the left and to the fifth on the right. This was followed by an area of analgesia ending at the seventh dorsal; also distinct tract sensory changes confined to the left thigh. There were also inconstant sensory disturbances along the anterior surface of the thighs from below Poupart's ligaments. There was a marked spastic paraplegia, and the patient was confined to a chair. Impotence and urinary incontinence were present. There were some slight trophic changes over the skin of both feet. The upper extremities, as well as the cranial nerves, were normal.

While at the Montefiore Home, the patient developed a sense of painful constriction around the chest. Coughing and

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sneezing increased the pain. The sensory disturbances are indicated in Chart 1. A diagnosis of spinal cord tumor somewhere between the fourth and sixth dorsal segments, situated at the right side of the cord, was made. Under these circumstances, another laminectomy was advised.

Jan. 2, 1914, Dr. Elsberg performed the operation. Following is his report:

Laminectomy and removal of extramedullary tumor at the fifth cervical segment. Typical removal of spinous processes and laminae of sixth and seventh dorsal and first dorsal vertebrae. Incision of dura. Small

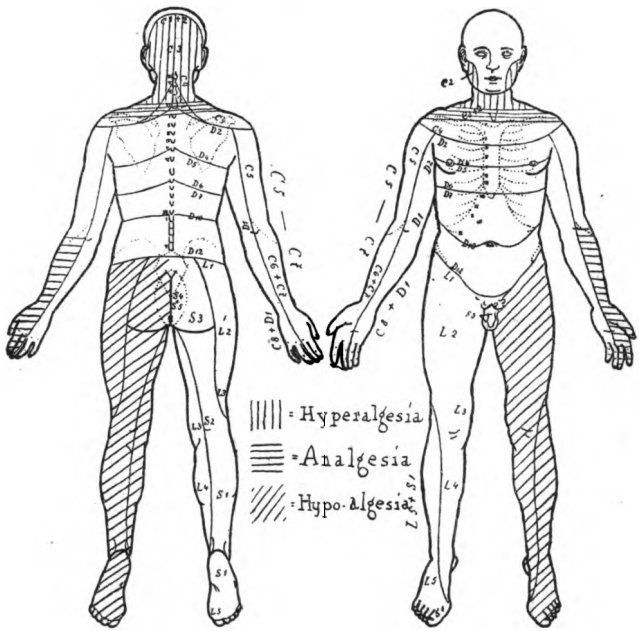

Chart 2.—Sensory disturbances in Case 2.

amount of cerebrospinal fluid escaped. No evidence of a new growth. With a probe, a resistance was felt about 2 inches higher up. The fourth and fifth arches were therefore removed and the dural incision; lying on the posterior surface of the cord a little to the right was a small brownish-red tumor about the size of an almond. The growth was adherent to the inner surface of the dura by a few line adhesions; after the division of which, the tumor was lifted out of its bed and removed. Closure of dura, muscles, fascia and skin. Pathologic report of tumor: Psammoma.

July 1, 1914, the patient was discharged from the Montefiore Home practically well.

Case 2.—J. B., admitted, July 22, 1919, discharged, Nov. 13, 1919, whose family and personal histories were negative, felt

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sharp, needle-like pains in the three inner fingers of the left hand eight months prior to entrance to Montefiore Hospital. There was loss of dexterity in these fingers. Two months later there was paresthesia in the left hip and then in the left leg. Stiffness and numbness of the left leg interfered with walking. The pain in the fingers ceased with the appearance of stiffness in the left upper extremity all through the disease. The last symptom to develop was difficulty in starting the urinary stream.

Examination revealed: nystagmus to the right and upward; convergent strabismus in the right eye; angulation of both hands; wasting of the musculature of the left upper extremity, most marked in those muscles supplied by the ulnar nerve; brachialis anticus not affected; sagging of the left shoulder; von Grafe's sign of the left eye; the left pupil smaller than the right; reflex of upper extremities increased, left more than right; twitchings of the muscles of the left lower extremity; spasticity with ankle clonus of the left lower extremity; no Babinski reflex; xanthochromia with massive coagulation present.

Following the lumbar puncture, the level signs became more pronounced. The sensory disturbances are indicated in Chart 2. A diagnosis of a cervical cord tumor of the soft, giant variety was made. The highest level of the tumor was placed at the third cervical segment.

Sept. 18, 1919, Dr. Harold Neuhof performed the operation. Following is his report:

> Typical laminectomy incision. Spines and arches of fourth, fifth and sixth cervical and subsequently third and seventh; dura opened between traction sutures. There extruded at once, with an escape of some fluid, the upper cystic pole of the tumor. Posterior roots could not be found, and for some time it was impossible to determine whether or not the tumor was an intramedullary one which had broken through, and finally by carefully lifting the cystic cap the extreme upper pole slipped out from under the area of the second cervical and immediately after the lower pole, and then the whole tumor slipped free. Its attachments were two left posterior roots (left). Tumor arose from pia-arachnoid. The cord was extensively flattened, more so on the left than on the right side.

Case 3.—F. G., a woman aged 20, who entered the Montefiore Home, April 18, 1919, and who is at present in the hospital, presented negative personal and family histories, and gave the following chronological record of her symptoms, which began ten months prior to entrance: (1) diplopia; (2) weakness of the left upper extremity; (3) sudden pain in the back of the neck, radiating into the back of the head; (4) evidence of thermesthesia over the left shoulder; (5) weakness of the right upper extremity; (6) weakness of the right lower extremity; (7) loss of bladder control, and (8) quadriplegia.

It took ten months for these symptoms to develop. April 20, the right pupil was larger than the left. There was nystagmus to the right. The third and fourth cervical ver-

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tebrae were prominent and tender. There was marked atrophy of the entire musculature of both upper extremities, the left more than the right. There was reaction of degeneration in the intrinsic muscles of both hands. The hands were cold and cyanosed. The muscles of the back were weak. The abdominal reflexes were absent. There was spastic paraplegia with double Babinski reflex and double clonus. There was bladder incontinence. Marked trophic changes of the skin of both lower extremities had occurred.

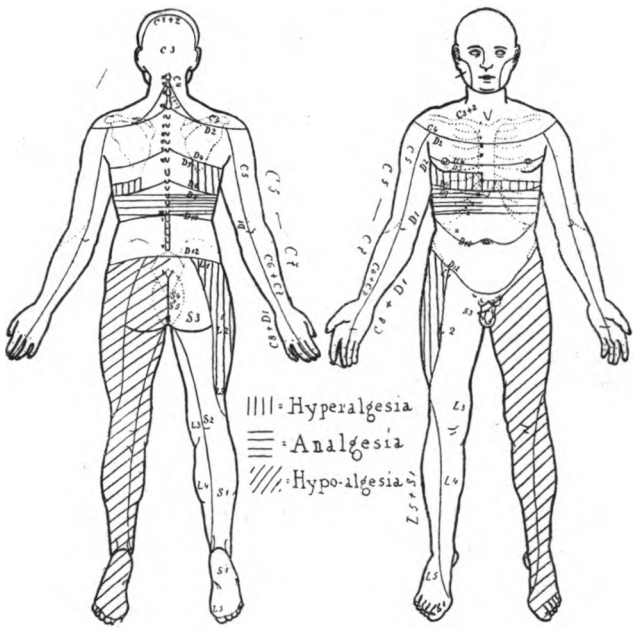

Chart 3.—Sensory disturbances in Case 5.

There was a decubitus ulcer over the sacrum. No disturbances of the thoracic and abdominal viscera were observed. Xanthochromia with massive coagulation was present. All the other laboratory examinations were negative. In view of the marked sensory and motor tract changes, as well as the rapidity of development of bilateral symptoms, a diagnosis of an intramedullary disturbance was made. Dr. Elsberg performed laminectomy of the third to the fifth cervical laminae. Evidence of diffuse intramedullary growth was presented.

Case 4.—R. S., aged 22, single, no occupation, admitted Oct. 23, 1919, died Jan. 1, 1919, whose family history was

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negative and whose personal history showed some traumatism of an indefinite nature, dated the present illness four years prior to examination. There was a pain in the back radiating into both lower extremities. Three years later thermesthesia was evidenced by a painless burn. There was progressive weakness of both lower extremities, and paralysis of both lower extremities. There were bedsores and urinary incontinence. (The exact condition at this time is not known, as the patient was in a different hospital.) Operation was performed at a different hospital. The report of the operation was: Lower dorsal and lumbar cord distended by an intramedullary cyst which, on operation, showed yellow fluid.

On entrance to the Montefiore Home, the patient showed a complete paraplegia with loss of reflexes and loss of all sensation from the umbilicus downward. There was an enormous bedsore all over the buttocks, with profuse purulent discharge of a very bad odor. The patient died, January 1, from general sepsis.

Case 5.—R. B., a woman, aged 37, married, admitted, May 12, 1916, left, Nov. 16, 1916 whose family and personal histories were negative, reported that the present illness began fifteen years prior with pain in the upper quadrant of the right abdomen. A diagnosis of gallstones was made, for which the patient was operated on without relief. Two years later an operation for appendicitis was performed, but no relief followed. She was next told that she had a femoral hernia and should be operated on. This time one of us was consulted. Evidence was found of level signs as well as motor tract interference affecting the right lower extremity. This disturbance, although not fully developed, yet showed evidence of a lateral tract involvement. The sensory disturbances are shown in Chart 3.

A diagnosis of an extramedullary spinal cord tumor on the right side of the cord and situated at the seventh dorsal segment was made.

Laminectomy for the removal of extramedullary tumors at the level of the seventh and eighth dorsal segments was performed at Mount Sinai Hospital, by Dr. Elsberg, Oct. 8, 1915. The arches of the fifth, sixth, seventh, eighth and ninth dorsal vertebrae were removed. The dura was incised. Considerable cerebrospinal fluid escaped. In front of the seventh and eighth dorsal roots on the right side were two small tumors connected by a fibrous band. Each tumor was the size of a hazelnut, and lay under but was not adherent to the posterior nerve root. The spinal cord was pushed backward to the left. The lower tumor was easily pulled out from underneath the eighth root, but the seventh dorsal root had to be divided before the upper tumor could be removed. The dura, muscles, fascia and skin were sutured. The tumors

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measured 1 by 1 cm. in size. The pathologic report was: fibroma.

Five months later the patient returned to the Montefiore Home with the history that for all this time she had felt comparatively free of all the symptoms. For the last week a complete spastic paraplegia developed with symptoms more marked on the right side than on the left. The sensory disturbances were more severe than before the operation; the level was higher than the site of the operation. Possibly

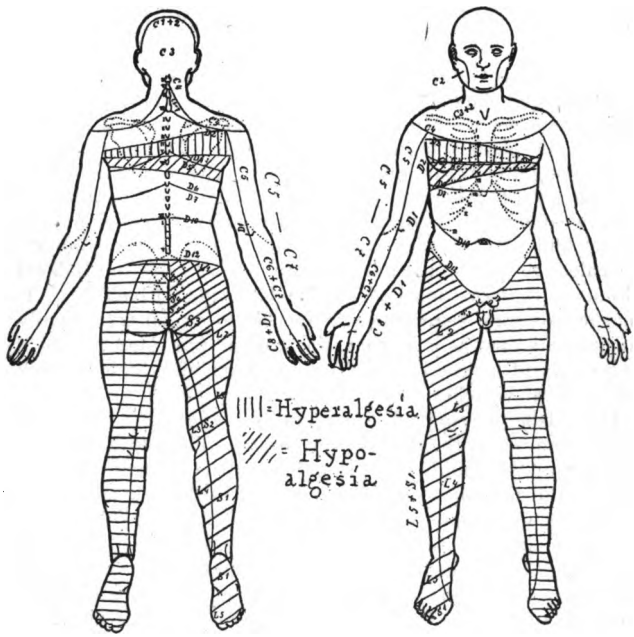

Chart 4.—Sensory disturbances in Case 6.

another tumor higher up was developed. The patient refused another operation, and left the institution shortly afterward with the condition unchanged.

Case 6.—G. W., a woman, aged 45, widow, admitted, Aug. 21, 1919, and at present still in the hospital, dated her illness from one year prior to her entrance to the Montefiore Home. The chronology of symptoms was: (1) pain in back of the right chest; (2) pain and burning sensation in the epigastrium; (3) increase of pain by coughing and sneezing; (4) weakness of the right lower extremity; (5) disturbance of gait, and (6) difficulty in starting the urinary stream.

Examination revealed a spastic paraplegia with symptoms more marked on the right side. There was a right Babinski

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reflex, right ankle clonus, etc. Both abdominal and cremasteric reflexes were absent. Xanthochromia with massive coagulation was present.

Following the lumbar puncture, the Brown-Séquard syndrome became more pronounced, and the level signs more marked. The sensory disturbances are shown in Chart 4. A diagnosis was made of an extramedullary soft tumor situated on the right side of the cord with its highest level at the third dorsal segment.

Operation by Dr. Elsberg revealed an endothelioma on the right side of the cord between the third and fifth dorsal segments.

Case 7.—G. G., aged 62, married, who was admitted, April 10, 1917, and died, Dec. 25, 1918, and whose family and personal histories were negative, gave this chronology of symptoms: (1) painful cramps in both legs; (2) severe burning pain along the right lower extremity; (3) dragging pain in the back; (4) tingling sensation in the soles of the feet; (5) weakness in both lower extremities; (6) inability to walk, and (7) bladder incontinence. Physical examination revealed a spastic paraplegia with signs, such as Babinski, clonus, etc., more marked on the left side (Chart 5). The marked sensory disturbances referable to dorsal tract involvement made us believe that the tumor was situated at the anterolateral aspect of the cord, with its highest level at the fifth dorsal vertebra.

Dr. Elsberg removed the spinous processes of the fifth, sixth and seventh dorsal vertebrae. The dura appeared whitish in the middle of the exposed part, bluish above and below, and by palpation a hard area could be felt through the dura. It was supposed that the hard mass was the tumor; but after incision of the dura, it was found to be the cord itself pushed backward and to the right by a small tumor which lay on the left side and on the front of the cord anterior to a slip of dentate ligament. A tumor the size of an almond was found on the anterolateral aspect of the cord, which was very slightly adherent to the dura and which was easily removed.

From the report of the operation, it is easy to explain the occurrence of the dorsal tract symptoms. Pathologic examination of the tumor demonstrated that it was a fibrolipoma. The patient made an uneventful recovery and was free from symptoms for about a year. At the end of two years the patient returned with these symptoms: (1) severe pains in both legs; (2) weakness of the lower extremities; (3) inability to walk; (4) severe girdle pains, and (5) urinary incontinence. There was complete spastic paraplegia with large decubitus ulcers. Gangrene of both lower extremities soon set in, and was followed by death from general sepsis. No necropsy was performed.

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CASE 8.—L. B., a man, aged 24, single, tailor, admitted, June 15, 1915, discharged, May 1, 1919, much improved—practically well—whose family and personal histories were negative, had been ill fifteen months. The illness began with pain in the right shoulder radiating into the neck and right upper extremity. There were cramps in the right upper extremities, and pain in the left upper extremity. Paralysis of the right upper extremity was followed by paralysis of the left upper extremity. There was gradual paralysis of both lower extremities, preceded by retention of urine. Twitchings occurred in both lower extremities. Roentgenos-

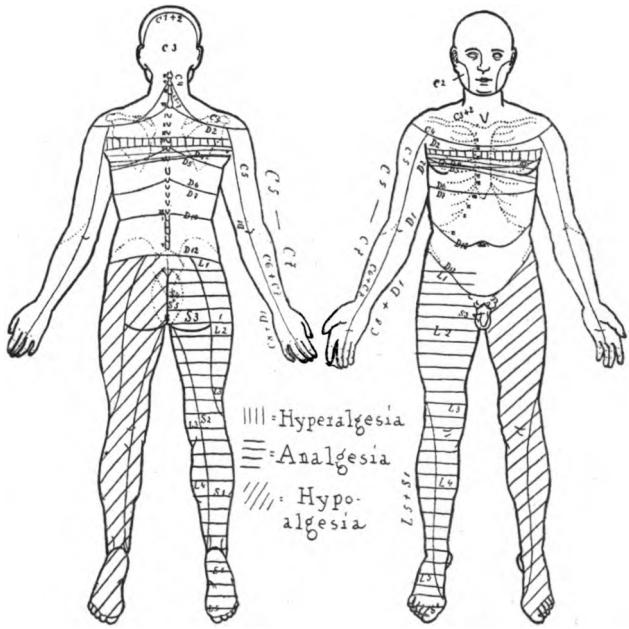

Chart 5.—Sensory disturbances in Case 7.

copy revealed a moderate degree of arthritis of the third, fourth and fifth cervical vertebrae. Physical examination revealed spastic paraplegia with symptoms more marked on the right side and flaccid paralysis of the upper extremities. The sensory disturbances are shown in Chart 6. After prolonged and careful observation at the Mount Sinai Hospital, a diagnosis of cervical spinal cord tumor situated high in the cord was made.

An operation was performed by Dr. Elsberg at the Mount Sinai Hospital, who removed the spinous processes and laminae of the fourth, fifth and sixth cervical vertebrae, and

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incised the dura. When the dural sac was opened, it was noted that no cerebrospinal fluid escaped. In the uppermost part of the wound, inside the dura, was seen the lower end of a neoplasm which lay on the posterior surface of the cord a little to the right side. Below the tumor especially on the right side, the pia covering the cord was thickened and reddish-brown. In order to expose the upper end of the growth the arches of the second and third cervical vertebrae were removed and the dural incision extended upward; but even this did not expose the upper end of the growth, which must have extended up to the foramen magnum. The tumor was not adherent to the pia. The lower end was raised, and by gentle traction the growth was pulled out of the spinal column and removed. From the length of the tumor, it had evidently extended up into the foramen magnum. The exposed cord was much flattened. The pathologic report was spindle cell sarcoma.

Case 9.—E. G., a woman, aged 52, married, who entered, March 31, 1913, and died, June 6, 1914, and whose family and personal histories were negative, dated her present illness thirteen months prior to entrance. It began with severe bitemporal headaches. Eight months later there was weakness in the right arm. Ten months later there was weakness of the lower right extremity. Twelve months later there was weakness in the left upper extremity. There followed severe precordial pains, fainting spells, distention and swelling of the right lower extremity, and urinary incontinence.

The heart was enlarged to the left. There was right ankle clonus. The left plantar reflex consisted of an extension of the four outer toes, and flexion of the great toe. There was flaccid paralysis of the right upper extremity. There was no reaction of degeneration. The left upper extremity was weak, but not to such an extent as the right. There was marked hypertonia in the right lower extremity, with Babinski reflex. There were trophic changes of the skin in both lower extremities. The sensory changes are shown in Chart 7. A diagnosis of an intramedullary cervical tumor was made. Owing to the marked cardiac trouble, exploratory operative procedure was not undertaken.

At the necropsy, the brain and coverings were found to be negative.

Spinal Cord: The cervical cord was enlarged throughout its entire length to about twice its normal thickness, the enlargement being caused by the growth which occupied the posterior half of the cord and replaced the commissures, central canal part of the anterior horns, and the posterior horns and columns, more so toward the right side. The growth was grayish, soft and well defined from the surrounding structures, and there was no evidence of cavity formation. The growth extended throughout the entire cervical cord, but was more centrally located in the segments.

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Histology: The mass of what corresponded to the new growth seen microscopically was merely broken down material and was structureless. Peripherally, especially posteriorly, a narrow strip was found containing peculiar darkly staining cells of varying sizes and shapes showing mitoses and frequently arranged in columns. Here and there were giant multinuclear cells. The pia was much thickened posteriorly and laterally, and the cells described also were found in the

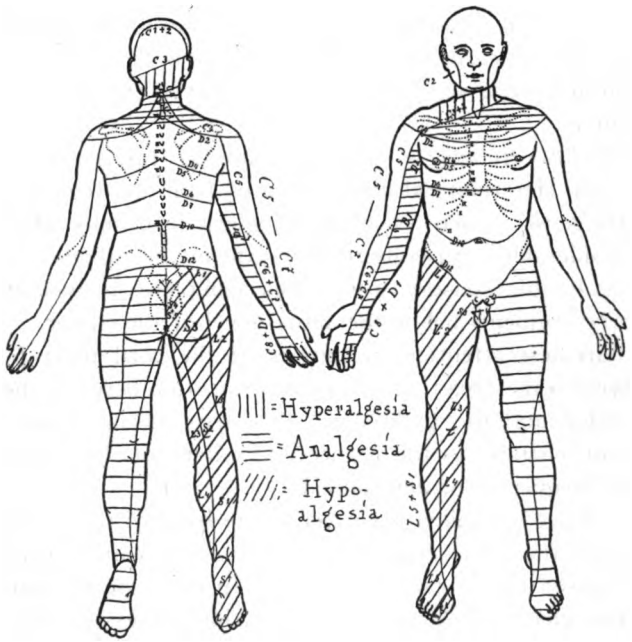

Chart 6.—Sensory disturbances in Case 8.

pia, which was intimately connected with the cord. The blood vessels were much sclerosed, and many ganglion cells of the anterior horns showed various stages of degeneration. The diagnosis was gliosarcoma.

SIGNS POINTING TO SITE OF TUMOR

From our study of spinal cord tumors, we could deduce the following signs as a guide for the establishment of the actual site of the tumor:

1. *Valuable Signs.*—(*a*) Root signs; which consist of neuralgic pains with symptoms pointing to a distal distribution of an affected nerve.

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(*b*) Zones of hyperesthesia immediately above the seat of the tumor and more marked at the homolateral site.

(*c*) Level abolition of skin and tendon reflexes.

(*d*) Deep spinal tenderness.

(*e*) Alteration in vibratory sense.

(*f*) No sweating below the level after injection.

(*g*) Ocular symptoms, such as lateral nystagmus and difference in the pupillary size as well as the palpebral fissure, which may all be present in high cervical tumors.

2. *Less Reliable.*—(*a*) Homolateral paralysis.

(*b*) Heterolateral distribution of pain and temperature sense, which reach their highest level only after considerable cord compression, late in the disease.

(*c*) This sensory loss, when complete, is usually three segments below the actual level of the tumor.

Greatest attention in tumors of the cord must be given to the area that marks the transition between the normal and the pathologic; this area merits a most careful study, as the nature and extent of the tumor can frequently be determined.

What has taken place below the level of the lesion where the compression is at all marked can only be surmised in the rarest of cases. In one case of multiple tumors of the cord, two affecting successive roots could by post facto study have been determined. These were two successive root zones involved according to the history of the case. As to the occurrence of xanthochromia, we have nothing to add to what has already been said. In our experience, the Froin reaction was of great diagnostic value, indicating interference with the free circulation of the cerebrospinal fluid.

As to the results of the operation: In two cases, the tumors recurred; in one case, multiple fibromas after five months of comparative well being; in the other case, a fibrolipoma after two years of comparative well

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being. In only one case, the intramedullary cyst, do we believe that the condition was possibly aggravated by the operation. In other cases, gratifying results were obtained. Of course, the best results were obtained in the soft tumors. In one case, the patient was up and about three weeks after operation, and within four months was able to return to work.

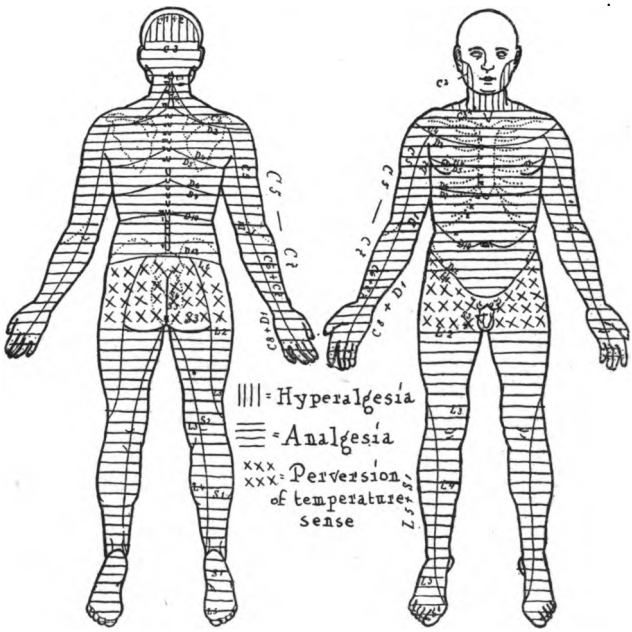

Chart 7.—Sensory disturbances in Case 9.

CONCLUSIONS

1. Progressive spinal cord diseases giving level signs and symptoms should be carefully observed and studied.

2. When no distinct level can be established, the effect of lumbar puncture should be carefully watched.

3. Xanthochromia of the spinal fluid in level spinal cord progressive affections usually means spinal cord tumors.

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4. One should operate only when a level has been established. Probing above and below the suspected site, while useful in many cases, often fails.

5. Operations, frequently by their decompressive effects, help even in intramedullary tumors.

6. The possibility of a soft tumor in atypical level cord lesions must be borne in mind, as these are most frequently overlooked.

ABSTRACT OF DISCUSSION

Dr. Julius Grinker, Chicago: Dr. Climenko has fallen into the error of giving us only classical descriptions, though I am sure he must have had cases that did not comply with the classical types and their differentiation into the so-called extramyeline and intramyeline symptom complexes. I have seen cases of extramyeline tumor without pain, and I am sure every one has at least, not early, when we expect neuralgiform pains. On the other hand, I have seen cases of intramedullary tumor in which there was pain. I am somewhat disappointed that Dr. Climenko did not tell us something about the syndrome of circumscribed serous meningitis. Circumscribed collections of serum in the soft membrane are capable of giving symptoms of spinal cord tumor, but there is a differential point in these cases worthy of emphasis, and that is, that the level of anesthesia is apt to vary from time to time. Dr. Climenko emphasized the point that after operation for spinal cord tumor we must not expect immediate results; a year may elapse before the first signs of improvement are noted. Whenever you have the feeling that your case is not one of multiple sclerosis, nor syphilis, advise an exploratory operation at the level of anesthesia, because if you only find a tumor in one out of a hundred suspected cases it has been worth while to do the operation.

Dr. Alfred W. Adson, Rochester, Minn.: I have seen quite a number of spinal cord tumors, and have found that one rarely finds the textbook picture. The history is probably the most valuable point in differentiating between chronic meningitis and serous meningitis. Spinal cord disease may have had a long or a short symptomatology. It has progressive motor disturbance, which may be more marked on one side than on the other, but it is slowly progressive. A man will discover that his sensations are changing, his ability to walk is getting less and less, and sooner or later, unless the cause is removed, he becomes helpless. Serous meningitis is the most confusing of all spinal cord lesions to differentiate from tumor. If in doubt, do an exploratory operation. But you must have a level. In 1917 I had sixteen cases with thirteen tumors demonstrated of which eight were removed,

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and three cases were serous meningitis due to arachnoiditis. As far as results are concerned, usually after the tumor has been removed one very soon sees improvement in the condition, provided the duration of the tumor has been short. Patients with a history covering a year or less usually recover completely, provided the tumor has been removed intact. But when complete paralysis has existed for more than one year much improvement cannot be expected.

Dr. Andrew L. Skoog, Kansas City, Mo.: I have never been able to differentiate the soft from the hard types of tumors. I doubt whether this can be done very often. The consistency of the tumor does not matter anyway, but it does matter whether it is a malignant tumor, and especially an intramedullary tumor, or a nonmalignant tumor. Most of the malignant tumors involving the medullary substance are inoperable from the standpoint of cure but the patient may be benefited. The extramedullary tumors should have a very high recovery rate. In view of the fact that we cannot always distinguish between intramedullary and extramedullary tumors, and malignant and nonmalignant tumors, it is necessary to operate in all these cases, and it is important to have a level. Undoubtedly a number of operations have been performed at too low a level. Sir William Gowers said: "When in doubt go even one or two levels higher." I have noticed xanthochromia in a number of cases of spinal cord tumors. It is an important part of the spinal cord tumor syndrome. However, xanthochromia can occur in other diseases.

Dr. Karl A. Menninger, Topeka, Kan.: What changes were observed in the colloidal gold chlorid curves in spinal cord tumor?

Dr. M. A. Bliss, St. Louis: The marked variability of symptoms in the solid tumors would lead one to believe that after a time the cord acquires a tolerance that enables it to resume its conductivity even in the case of a solid growth. We have demonstrated this more than once. Serous meningitis may involve a considerable length of the cord, therefore you may have a fairly well established level and evacuate a considerable accumulation of fluid, and then, even with a marked degree of recovery almost immediately after that evacuation, the condition recurs. Then, again, it may be associated with tumors in other places in the cord.

Dr. George A. Moleen, Denver: Was your case one of psammoma or was it a case of multiple tumor which probably developed from a process originating in the pineal body from which such tumors nearly always originate and with which they are closely associated?

Dr. Tom A. Williams, Washington, D. C.: The diagnosis is usually very easy, but we try to avoid waiting until there is a well established syndrome. Early diagnosis in cases of

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extramedullary growth is based largely on root signs. Root signs depend to a great degree on the stem localization and on a very delicate examination of the sensibility, which can be confused very easily with the signs noted in the rather rare cases of gliosis leading to syringomyelia in the posterior root zone. The hypalgesia and the hypothermy in the arms noted by Dr. Climenko must be dependent on group symptoms alone because the thermal conductivity crosses the cord only from six to eight segments above its entry to the root, and an interruption of thermal conductivity, therefore, denotes a growth six to eight segments above the region where it seems to be interrupted, unless the growth is on one side interrupting only the thermal conductivity fibers, in which case there are certain to be other symptoms referable to that lateral column of the spinal cord. The difficulty of diagnosing the early case is very great. The cases we have seen are all late cases, and the difficulties presented are those arising from differential diagnosis between a tumor and serous meningitis. Severe mental symptoms are quite uncommon as the result merely of stasis in the spinal fluid. In a protracted case with spinal cord symptoms and delirium, we should think of serous meningitis. Improvement in these cases is an important point to consider. I would like to know why these improvements occur. We have all seen remarkable improvement in cases of trauma in which the functions were completely suspended and actual injury of the cord producing Brown-Séquard syndrome has occurred, recovery taking place after several months. So we must seek more satisfactory solution of the question.

Dr. Hyman Climenko, New York: Dr. Grinker referred to spinal cord tumors in which pain is absent. We had no case without pain, therefore the point was not mentioned. The most difficult part of the examination is establishing a level, particularly when with a neurotic patient, and people of low intellectuality who cannot distinguish between hyperalgesia and hyperanalgesia; people who scream when suffering pain and keep on screaming when you get to an analgesic area. Therefore, you do not know whether you have passed a hyperanalgesic area or an area of normal sensibility. The establishment of a level is also difficult in tumors of the soft variety and in multiple sclerosis. But there is a differentiation between the two. The level of multiple sclerosis never remains permanent for any length of time. There is also a change of level in the soft tumor, but the change is not as marked as it might be in the other condition. As to the length of time necessary for recovery after operation: In our cases the time varied from four months to six years. In one case of the soft tumor variety, the patient was out of bed and comfortable within three weeks after the operation. Another point which should be emphasized is that pain after operation is usually greater than pain before operation. Of

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course we attempted to exclude syphilis, in all cases, but syphilis and spinal cord tumors may coexist. When there is a persistent level and there is no improvement in symptoms after specific treatment, operate in spite of the positive Wassermann. I agree with Dr. Adson that the history is very important. You do not always get a classical level or a classical picture, but it is the history of a slow and constant progression of symptoms that helps to make the diagnosis. As to distinguishing between the soft and hard tumors, so far as the results of operation are concerned it is not of much importance, but in diagnosis it is of great importance. It was the soft type that always gave us trouble in attempting to differentiate these cases from other spinal cord disease. We did not employ the colloidal gold test. The psammona patient has been entirely well since 1913. I do not believe the pineal gland was involved. From first reported the existence of xanthrochromia, not in connection with spinal cord tumors, but as related to inflammatory disease. I spoke of xanthrochromia as an important symptom in diagnosis, for whenever xanthrochromia coexists with a slowly progressive lesion of the spinal cord, a tumor should be suspected.

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THE CAUSES OF EMOTIVITY, AND THEIR MANAGEMENT

TOM A. WILLIAMS, M.D.
WASHINGTON, D. C.

It is now a commonplace that perturbations of emotion are frequent causes, not only of inefficiency, but also of ill health of the body itself. The physical consequences of emotion are well attested even in the laboratory. To the physician, this knowledge, while a satisfaction, is only a preliminary to the search for its application to the cure of disease. To this end he seeks, first, to recognize such diseases to differentiate them from others, and secondly, to remedy them. Recognition is difficult; for the presence of an emotive state is not enough to signify that the disorder is primarily psychogenic.

Many physical conditions are characterized by disturbances of feeling, emotion. Intoxications, many infections and visceral diseases all disturb the emotions, and often seriously. So do many diseases of the nervous system itself, such as cerebrospinal syphilis, disseminated sclerosis, neoplasms, and what are called the great psychoses. These are all disorders of somatic source, the emotional disturbances being incidental, and not susceptible of being dealt with fruitfully by psychologic means; for only the disappearance of a diseased bodily state will dissipate the morbid emotion in cases of this kind.

The emotions of psychologic origin are numerous. They are not confined, as some writers would have us believe, to sexuality. Not always are they the fruit of an internal conflict; not seldom are they quite independent of attempts to repress them.

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For instance, a woman was frequently and explicitly animated by an intense hatred of her mother-in-law which she made no attempt to repress or conceal. This aversion she frankly expressed to her husband without any assuaging of the emotion, or of the suffering it induced, although she recognized that for economic reasons it was impossible to split up the household.

Similarly of jealousy. In a number of patients no repression has been attempted, and the jealousy has not even been deplored.

Mortification is a very frequent disabling emotion. It is based on shame and is always due to an inadequate envisagement of the situation. This, in turn, has come about from imperfect knowledge of oneself, and this in turn has often been determined by circumstances giving a false perspective in relation to some mode of feeling or behavior. Quite often this has to do with sex material, but quite as often it is due to social causes and the inculcations of others in the family, in school, in business or in society. Intrinsically it is a fear mechanism; indeed, fear is by far the commonest of the incommoding emotions. When acting chronically it shows itself as anxiety, and is so frequent that some writers have given a special name to the state: "anxiety neurosis." The French have further differentiated, and long ago called "anxiété" the more psychic manifestations, giving the name of "angoisse" when circulatory and respiratory manifestations are conspicuous. It is the state which the Germans call "angst."

Not fear itself, but the feeling of impotence is sometimes the dominant emotion. This may arise from a feeling of incongruity in the situation so marked as to appear insoluble. In some temperaments, rebellion and struggle against this is quickly replaced by resignation. This is particularly true of the incongruity which gives rise to the feelings of disillusionment. This is an emotion most destructive to the employment of the full activity of the personality in work or in human relationships.

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It has been the fashion to say that the emotional aspect of all these situations is of transcendent importance in a psychotherapeutic sense. This error is only another instance of the very common one in medicine of being unduly swayed by the conspicuous presenting symptom. Surgeons have long ceased to depend for their judgments on the patient's idea of the relative importance of symptoms. To the psychoneurotic patient, the emotion is all important. To the discerning psychopathologist, the really important matter is the causative one. Now, an emotional situation is very rarely direct. One might almost say that it is invariably determined. It may safely be postulated that emotion occurs only when the subject perceives an occasion for it. The psychologic process is one dependent on sensation, perception, cognition—that is, intellection. This is so even in a rudimentary instance, as that of instinctive reactions of animals, as the scuttling of fish at the sound of a footfall, or a horse running in terror from a strange object.

The emotion is dependent on the perception. Physiologically, too, it may be stated that an emotion is always conditioned by a perception. This is just as true of emotions which appear autochthonous, endogenous. The emotion originally induced by a former perception is now induced by the recollection of it. However, through association another circumstance may supplement, or even be substituted in recollection for the original inducing circumstance, so that an idea apparently quite extraneous to the provocative one may seem at first sight quite incongruous with the emotion it seems to produce. The reaction is then adjudged as a morbid one although in reality it is a perfectly natural response to circumstance. The circumstance itself, indeed, in such cases is not merely what it appears. There has always been added to it an element unperceived by the uninitiated, which is the real determinant of the emotion produced in the subject.

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For instance, listening to a sermon in a crowded church would not seem to be sufficient to arouse the emotion of terror to such an extent that it would prevent the subject thereafter from going to church, theater, or crossing the street. It was only sufficient in virtue of its association with a faintness and oppression which had occurred on one occasion. The significance of this association had not been grasped by the patient. Indeed, the circumstance was a memory apart. It was only when the patient was made to grasp the real significance of this circumstance that her belief could be changed regarding situations which for eight years had been arousing terror and panic, and had brought her to such a degree of inefficiency that she could rarely bring herself to cross the street alone.[1]

Even in the dream in which the content seems to be under the sway of the general feeling tone of the moment, in reality the direction of that feeling tone, and the elaboration of the imagery are dependent on former associations, which of course are the fruits of cognition.

IMMEDIATE EMOTIONS

It is just as true that the material of brooding or rumination is very often determined, not intellectually, but by the feeling tone, and that the judgments are thereby vitiated. The recognition of this is so universal, it is such a commonplace, that we can scarcely wonder that psychopathologists have carried into their studies of emotional manifestations the induction that the feeling tone determines the thought, forgetting entirely that while this is true, both of depressions and exaltations, yet that in the very different states of besetment the reverse is the case. To specify, the common saying "to have a jaundiced view" represents the man whose feeling tone, disturbed by physical agents, permits him to harmonize only with depressing circumstances. The disordered metabolism of the constipated or the hyperproteotic disturbs the feelings, so that automatically unfavorable or depressing points of view are more easily adopted than cheerful ones.

1. Agoraphobia and Claustrophobia Removed in a Week, Philadelphia Neurological Association. A complete discussion of this case is given, International Clinics 4, 1919, under the title "Differentia of Phobias and Obsessions."

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The consumptive, whose metabolism is speeded up to produce reactions by which to cope with the invasions of the tubercle bacillus, finds his feeling tone stimulated and active; hence he is in harmony with cheerful, hope-giving situations, and passes by without notice the circumstances of depressing character. None of these reactions, nor the many others like them, are ratiocinative. They are purely instinctive and depend on autonomic states. They are not controllable directly by will. The impotence of psychotherapy in such situations is represented by the adage: "Never yet saw one a philosopher with the toothache." It is a truism in business not to expect a judicial decision the morning after a debauch.

THE MEDIATE OR INDUCED EMOTIONS

The contrary mechanism is so simply exemplified experimentally during the study of the secretory sequences of emotion that it is surprising that there are still those who fail to give it due significance. In all the experimental instances it is necessary to provoke the emotion studied by first inducing the cognition of the situation by the animal or the human being experimented on. But without any experimental instances we can again resort to common experience in studying the effect of bereavement or ruin of the mechanism which determines the stampede of a crowd, of the various methods of suggestion, such as the flowing of tears in witnessing a melodrama, or the indignation at the abominableness of the villain in a romance.

Such emotions as the anxiety so often contingent on the accomplishment of work under another, or the fear of losing one's position, or the fear of failure, when analyzed, will always be found to hinge on apperceptions.

No situation apperceived can be divorced from elements contributed by the percipient. To no two men is a given situation alike. This has been expressed in Hamlet by the remark: "There is nothing either

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good or bad, but thinking makes it so." It is, therefore, the patient's attitude of mind toward the situation that develops the emotion regarding it which disturbs him.

As a matter of fact, however, this is not autochthonous. It is invariably induced. The induction is usually performed by the mechanism of suggestion. This may occur at the moment itself through the word of another directly applied, as when the nurse says to a child, "Do not be afraid," thereby clothing the situation with the idea of danger, regarding which the emotion of fear is legitimate. The suggestion, however, is usually far less simple, and often depends on a logical induction, though of course always unaccompanied by full knowledge of true relationships. For instance, the so-called birth phantasies are frequently constructions of a child's imagination fabricating an explanation by conjoining, with the untruths he has been told, the facts he has observed.

Too frequently to the child are presented utterances in which error or mystery is implicit. Over and over again do psychopathologists find constructs arising logically from material in which the careless word of an elder did not recognize the implicit emotion for the child.

For instance, a feeling of impotence, and hence a diffident emotion regarding the subject of Latin, was the unfortunate heritage donated to a boy by the injudicious insistence of an elder brother that he was too stupid to study the classics. The fixed idea that resulted vitiated his life by turning him in an artistic direction, for which he was ill fitted, and preventing him from going into professional life, for which his scientific mind would have been admirably adapted.

A woman acquired a habit of mumbling because as a child she was ridiculed on account of the shape of her mouth and its size when she laughed. In the endeavor to minimize the conspicuousness she avoided opening the teeth widely, with extensive movements of the lips, thereby affecting the distinctness of her articulation.

Indeed, stammering is very frequently induced by dread of making an error of speech, or misstatement of fact, or of saying something that will arouse

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mockery. The induction of this emotion is a function of overstressing by parents or guardian, which acts through a mechanism involving in the speech what golfers call of stroke, "pressing." Having failed several times, especially after ridicule, the child is expectant of future failure and becomes ashamed of it. In consequence, he develops phobia at the prospect. The phobic state in turn induces such physical consequence as embarrassed respiration and tautening of phonatory muscles. This in turn may lead to the use of artificial devices in speech, which temporarily assist, but eventually have also to be got rid of when the stammering is dealt with.

THE PRINCIPLE

All this psychologizing would be superfluous were it not that it is absolutely essential for the clear understanding of the principle of the induction of emotion. It is by a grasp of this principle that the psychotherapeutist can best recondition his patients' reactions in such a way that circumstances which formerly induced distressing or incapacitating emotions shall no longer do so. This is most easily done by the patient when he is made to ascertain the component of the situation which really induces the fear. In consequence he can then imbue the situation with the feeling which to it is legitimate. This readjustment cannot always be effected at once. Practice may be required in concentrating on the efforts needed, for in some individuals the very attempt to consider the situation at all calls into play the very emotion which the whole process is designed to undermine. This, however, is merely a matter of habit, and with practice does not take long to overcome. Very often the reconditioning is precipitate, if not at the first interview, then as a culmination or explosion, so to speak, of a consistent effort during several days. It may take even longer.

Obsession in these cases is merely the haunting of the patient by an idea distasteful to him because of

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some emotion accompanying it. The distress experienced by these patients during their ruminations is a legitimate enough accompaniment of their failure to direct their thoughts into channels which they believe are more proper or useful. But for the emotional element of the obsession it would cease to beset.

Of the tics, which are impulsions, and of course psychogenic, the same thing is true. They are derived from a movement of purposive intent. The movement has been originally adopted either to avoid discomfort in the part moved, or in response to reactions toward psychologic discomfort.

Thus, one woman felt impelled to turn her head to the right and behind, because there sat a fellow worker with whom she urgently desired an explanation regarding a misunderstanding.

Another woman turned her head to the side away from which her niece used to walk because of the fact that this girl had become engaged to a foreigner, and the aunt was strongly imbued by the belief that marriages with foreigners turned out badly; this because of her experience with many in her family and among her friends. Hence, she could not bear to look at her niece on account of the apprehensive grief at the prospect of losing her to misfortune.

Of course, the patients were not aware of these reasons. They had to be sought.

MANAGEMENT

The treatment of morbid emotions of this kind, then, depends on the finding of the situation, the prospect of which determines the emotion. It is with the cognitive element of this that we must deal by persuasion. Although we say that we change the mental attitude of the patient, we in reality change the situation itself by adding to it or subtracting from it the elements which arouse the incommoding emotion.

To do this is an art, and of course requires practice; but no amount of practice is of value unless the mind of the operator is directed toward the proper steps to be taken in performing his physiologic operations.

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EXTRAMURAL METHODS

It is true that similar reconditioning of mental attitudes and changing of emotions have always been accomplished by means of religion, philosophy, through charlatans, and by accident; but these agencies all possess serious inconveniences. The modification of the patient's religious belief is not the legitimate function of a physician. The practice of psychotherapy by priests is ineffective, as a rule, because they have not acquired the necessary knowledge of neurology, without which errors of diagnosis are inevitable; nor do they know the kind of psychology necessary for psychotherapeutic reconstruction. Indeed, this kind of psychology requires as an essential basis a sound knowledge of physiology, as well as a very thorough knowledge of clinical psychopathology.

Thus, a sea captain was referred because of distressing facial tics which prevented him opening the eyes at will, so that he could not go about alone without great distress, and could not read at all. The tic was the product of the anxiety induced by intense naval responsibility during the war. The systolic blood pressure was found to be 107, and the diastolic, 63. This I attributed to depleted endocrines. Suprarenal substance quickly raised it to 152 systolic, 108 diastolic, more in conformity with the state of stress at his age, 50. The stoppage of suprarenal substance and reduction of protein then permitted a fall to his norm, systolic 125, diastolic 90, the psychotherapy that cured the tic having also permitted recuperation from exhaustion.

Laymen are not trained to make differentiations of this kind, and the need for them is constant in the practice of medicine, to which the psychologic branch is no exception.

The same reasons make dangerous the work of philosophers and psychologists with these patients. It is still more true of the pseudoscientific modern religious movements which have healing as a function. In one of them, indeed, a destructive negativism is the keystone of the treatment. If the patient can be made to believe that no situation is evil, that which has

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aroused his inconvenient emotions necessarily ceases to do so; but the negation of reality is far too heavy a price to pay for the dispersal of an inconvenient emotion. The remedy is worse than the disease.

Thus, exhaustion is not only a cause of emotivity; but in some instances it is caused by prolonged emotions. The intricateness of these mechanisms is one of the vicious circles so much spoken of many years ago. Proper delving into genesis eliminates much of the imprecision attached to these cases.

The occasional removal of emotion by charlatans is effected by suggestions usually masked in physical measures, which then act by chance in accordance with the principles set forth in this presentation, namely, the reconditioning of the patient's attitude toward the situation. But when the charlatan does so, it is by accident, so to speak, and without the least knowledge of the great principle at work; hence he very frequently misses fire.

Accidents are sometimes efficient.

Such was the case of the man who had not walked for seven years, after a street car accident, for which he was heavily indemnified, until one night after the plaster fell from the roof his wife found him sitting in the chair in the corner of the room trembling with fright. Thereupon she induced him to make the necessary efforts to stand and walk, the success of which dissipated his phobia regarding his power of maintaining equilibrium.

As against the numerous failures of any of these methods, the almost universal success of the sound method of rational persuasion forms a striking contrast. My personal experience of twelve years shows that it is quite unusual that a morbid emotion cannot be dispelled and permanently so, and that the time taken to do so is more often less than a week than more than a month. The cure of emotions in military life, as during the great war, shows that these pretentions are well justified, and that such success should be the rule and not the exception.

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ABSTRACT OF DISCUSSION

Dr. Julius Grinker, Chicago: There is no doubt that Freudism is responsible for a flood of publications of the type that Dr. Williams has given us today, and many of these papers differ from each other only in that they use another phraseology. To practice neurology successfully, we must follow the various methods of psychotherapy and choose the method that will suit the individual case. A neurologist who knows how to get into the psychology of the neurotic will be able in most instances to get at etiologic factors without much trouble. But this alone does not cure the patient. It is a question largely of the susceptibility of the patient to suggestion, and no matter how I may struggle against accepting such a view, I think after all the successful treatment of the neuroses is due entirely to suggestion.

Dr. David S. Booth, St. Louis: I have not been able to get away from the physical basis of the psychoses. I believe that the fundamental element in the production of these psychoneuroses is in the anatomy or physiology or histology, whichever one may appear in a given case. I have not yet accepted the theory that these conditions are caused by suggestion or that they are cured by suggestion entirely. The fact that many of these patients recognize the error of their obsessions shows that they cannot be cured, in all cases at any rate, by suggestion. Such cases show that the disease is not caused by the idea, but by some physical condition of the body. The basis is there, and the idea is the result of this anomaly. Faith cure, or psychotherapy, if you want to give it a name that will be accepted by the medical profession, simply gives these people a settled philosophy of life. If their condition is not severe, by which I mean if the changes in the nervous system are not great, they will, perhaps, recover from that treatment alone. But I believe that in all cases the physical basis of the condition should be treated, at least in conjunction with the mental element.

Dr. Hyman Climenko, New York: There are distinct psychic traumas, distinct psychic conditions, that produce neuroses, and these neuroses are cured by psychic methods, whether Freudian or Christian science or rabbinical, etc., does not matter. The Freudians have contributed largely to the study of psychology, although they have not in the matter of treatment and cure. I know of many cases in which psychanalysis has aggravated the condition. Dr. Williams mentioned high blood pressure. This is often produced by psychic trauma, in which case, the high blood pressure will be reduced simply by talking with the patient and appealing to his reason, in other words, using one or the other type of psychotherapy.

Dr. Karl A. Menninger, Topeka, Kan.: There is constant talk about curing patients by "persuasion," by "suggestion,

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by "explanation," by "analysis" but by what particular technic is rarely indicated. It is rarely clear whose method is used or what particular steps are taken; consequently these men only encourage the skepticism of those whom they would convert. I suggest that they come to this meeting and stage an impromptu session of this kind; have a voluntary patient from among ourselves come forward and be treated by the method of the author's choice, here in front of us all. Then all may see what the psychotherapists do instead of hearing about persuasion or reeducation or readjustment by name only which never gives much to some of us because we do not know what the psychotherapist does when he does it, or what he means when he says it.

Dr. C. F. New, Indianapolis: In regard to the psychogenic origin of the underlying ideas of anxiety, which is the basis of the majority of the fears which beset these patients: A close analysis of a large percentage of these conditions will disclose the fact that there is an underlying source of worry which the patient is trying to overcome and which he does not want any one else to know about, and until that phase of the condition is cleared up or it is suppressed there is no peace of mind for that individual, no matter what you do. Before any psychotherapist or anybody else can make any impression, he must secure the confidence of the patient, and the best way to do this is by detailed, careful examination of the patient, his history and his present condition.

Dr. Ross Moore, Los Angeles: One neurologist will say that it is impossible to make these people better, while another will say that a great deal can be done by psychotherapy. Both are right, although seemingly opposed to each other, and the reason is this: One man has in mind the classification which refers to the individual whom I call the essential inadequate—the individual who is narrow and cannot be otherwise.

Dr. G. H. Benton, Miami, Fla.: Even here, before a body of neurologists and psychiatrists, the personal reaction to the words psychanalysis and psychotherapeutics tends to bring about at once a very marked personal equation, individual in character, and of emotive quality. This is the very thing that is occurring continually in psychoneurotic patients who are very limited in their field of apperception. Therefore, they lack judgment. Their emotivity has a great range and is very vacillating, but the means which we use to reach them, or in other words, the tools we use, remind me very much of our automobiles. They are selected by virtue of our particular choice, but we all "get there" just the same. And so it is with the neuroses. We accomplish results first, by our own understanding of the case, through our own methods, by our own individual possibilities, by first determining what exists and then by inquiring of the patient

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what does this mean to you, what does that mean to you, what does this situation lead to, and how do you react to it, and, then, by trying to interpret these things, and to determine further if the patient actually understands the words we use as we understand them. We often observe ourselves jumping around from one theory to another because it does not quite appeal to us or because we can find methods by which we can reach our patients and help them in spite of the individual equation—the patient himself. The personal equation is one of the greatest difficulties we have to contend with. This involves the personal equation of the physician as well as the personal equation of the patient. We are dealing with these things in our hospitals for the psychoneurotics. We are able to reach them to a greater or lesser extent but through very varied methods. Some are brought back to their former mental and physical level, others somewhat above that level. Although sometimes an immense amount of time is spent to attain the results and to insure their permeability, we are getting at it in our own individual ways.

Dr. Gustave Roussy, Paris, France (Translation by Dr. Williams): Babinski has long maintained that emotion is only an accessory or adjunct factor in these cases, the real factor being the suggestion, and that the emotion quickly disappears when counter suggestions are made. On the other hand, Dr. Roussy and his colleagues, along with Professor Dejerine, contend that the emotion is rather the primitive factor, because people are constitutionally or otherwise over-emotional, and they react in this way to the occasions which arouse the emotion. They think that their experience in the war confirms this opinion, while Babinski and his followers believe that the war confirms theirs.

Dr. George A. Moleen, Denver: I think we are privileged to speak of the neuroses as attributable to high elevations, since we find that the susceptibility to this emotional shock, to suggestion, to the other irritable influences, disappears when these persons seek the lower levels, such as the seashore. Some persons cannot live in the high altitudes because of the nervousness induced thereby. They could go to the seashore and become quite relieved and comparatively calm. In the war department manual is given a very clear description of the increase in nervous excitability experienced in high elevations. This, as I pointed out sometime ago, is due in large measure to the lowering of the blood values, the cellular and chemical elements, particularly the hemoglobin. Of course, we recognize the importance of constitutional inferiority and the relation of this constitutional inferiority to the endocrines as well as the conditions on which these high degrees of suggestibility may be premised. Not only do we have disturbances due to the hemoglobin

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and corpuscular diminution which was observed by Haldane, Barcroft and the men who headed the Anglo-American Pike's Peak expedition establishing the data with reference to the blood, but we can also look to the calcium deficiency as reducing the stabilizing character of the protoplasm so far as irritability is concerned. I do not believe that we should rely too strongly on the external influences, although they have their exciting possibilities, but we must also keep in mind the somatic disturbances and those forces which relate particularly to the body metabolism.

Dr. Tom A. Williams, Washington, D. C.: In my paper I adequately took care of all the physical causes which some speakers have brought forward. Such conditions as essential inadequacy or constitutional psychopathy we are not considering. What Dr. Neu said is justified—we must make the patient cure himself. When dealing with an inadequate you cannot expect of him what you would expect of a normal man. I deal here therapeutically only with cases in which the induction is purely psychogenic. In certain physical states the emotivity is exalted. In hyperthyroidism, for instance, all these factors are highly exalted; you cannot expect to cure that patient's emotivity by psychotherapy. We can only carry these cases along and help the physical treatment. Babinski has helped us to differentiate between organic and functional nervous disease. The differentiation depends largely on the anamneses and partly on the results of treatment. I tried to emphasize that all the emotive reactions of the kind of cases I speak of are purely secondary to the conditioning of an apperception, just as the animal which reacts from the emotive standpoint does so simply because of the apperception of the bell and the meaning attached to bells. As to the challenge to demonstrate a particular method, I am willing to do it, provided the right kind of case is selected, and inasmuch as certain patients have difficulty in doing this before audiences we must have privacy; and a patient should be chosen in whom a rapid cure is possible, and there are many. In the long standing cases that does not suffice: we must penetrate to the foundations of this thing, find the mechanism and reorientate the patient. It takes longer and one has to be familiar with psychopathic disease. It is not a matter of phraseology, it is a matter of principle. Suggestion must be religiously avoided. It is not upbuilding.

We do not need to interpret these things by dualistic philosophy. It is a monistic phenomenon purely. Psychologic reactions are physical reactions in reality, stated, however, dynamically and qualitatively only, as against those of the chemist which are stated ponderately, and those of the physicist which are stated quantitatively.

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MEASLES: BRAIN COMPLICATIONS

A. L. SKOOG, M.D.
KANSAS CITY, MO.

Even in a discussion of any neural complications or sequelae that might occur in a case of measles, we are compelled to admit and even bear in mind that the exact cause of measles still remains unknown. If the disease is studied from the standpoint of the epidemiologist or bacteriologist, the logical conclusion is that the etiology can be attributed only to some organism as yet unseen and uncultivated. Thus many investigators finally conclude that some ultramicroscopic organism is responsible.

We are forced to the conclusion that complications or sequelae, whether involving the brain, spinal cord or peripheral nerves, are uncommon. Yet they are probably of sufficient frequency, and certainly of sufficient gravity, to warrant a serious consideration. Surely we have no right to consider measles a simple and harmless affection.

Relative to the frequency of the neural disorders, I find that the recent literature bearing on this subject is not much more abundant than the ancient. According to Orthalan,[1] palsies following acute fevers were mentioned by Hippocrates and Galen; but the first allusion to measles in this connection was made by Olier in 1772, who referred to this disease as "a cause of acute hydrocephalus." H. B. Allyn[2] gives James Lucas credit for reporting the first case, in 1790, in the *London Medical Journal*. Of fifty patients seen in one epidemic in 1890 by Espagne,[3] one died from a complicating meningitis with convulsions and lethargy.

1. Orthalan: Bordeaux thèse, November, 1894.
2. Allyn, H. B.: Med News 59: 617, 1891.
3. Espagne: Montpellier méd., Series 2, 15, 1890.

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Wilhelm Möller [4] reports on 537 deaths in an epidemic of 1887, this being a death rate of 3.72 per cent. Among the many complications, convulsions and aphasias were rare. There were five cases of meningitis, three being reported as simple and two as tuberculous meningitis. A few of the textbooks on nervous diseases and works on internal medicine mention in a line or two the brain disorders resulting from measles, but many make no record of the subject.

A study of the literature indicates that the brain complications and sequelae of measles occur much more frequently in children than in adults, and during the convalescence much oftener than during the febrile or exanthem stage. Thus a large percentage of the disorders might be classed preferably as sequelae than as complications.

A large variety of pathologic lesions is reported as involving the central nervous system during and following measles. However, most of the cases found in the literature may be grouped under three or four headings. The meningitis group has a larger number than any other. Thomas [5] cites cases with meningitis, meningo-encephalitis, hydrocephaly, cerebral thrombosis, cerebrospinal affections, tuberculous meningitis, hemiplegia, apoplexy, hyperemia of the brain, and some others. Jürgensen [6] would doubt the accuracy of the Thomas reports, questioning often the responsibility of the measles.

I am reporting two cases which may be classed as sequelae to measles, one a cerebellar and the other a meningeal involvement.

REPORT OF CASES

CASE 1.—*A cerebellar sequela.—History.*—W. L. W., a girl, aged 4 years, whose mother was well and had had no other pregnancies, and whose family and past history was negative. had always been a robust, healthy child. Teething, walking

4. Möller, Wilhelm: Wurzburg thesis, July, 1896.
5. Thomas, Measles, Neural Complications, in Ziemssen: Cyclopedia of the Practice of Medicine, 1875, p. 97.
6. Jürgensen, Measles, in Nothnagel: Encyclopedia of Practical Medicine, p. 330.

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and talking occurred at the usual age. Measles was first determined by a typical macular eruption, Koplik spots, photophobia and other manifestations, Jan. 30, 1915. Eruption and acute symptoms were practically absent on the seventh day. About the twelfth day and during convalescence the mother noticed that the child did not use her hands quite the same, there being an unsteadiness when playing with toys or performing any voluntary act. The same state was seen soon in the lower extremities so that walking was impaired, and in a very short time she could hardly get about. There was an increased irritability, and some crying. Appetite and stools were normal.

Examination.—On the third day following the first neurologic manifestations, the general nutrition was fair. The temperature and the pulse were normal. There was a slight evidence of a past macular eruption. Cerebration was fairly good. The patient was inclined to be a little sulky or obstinate. The tongue was protruded mesially, but with much coarse tremor. There was a tendency to some nystagmoid movements when looking to the extreme right or left. Other cranial nerve findings were normal. The arms and legs had fair power for individual movements, there being no true palsies. Sensory tests gave normal responses. All deep reflexes were slightly increased, the right equaling the left. The superficial reflexes were normal. The movements in all the extremities were markedly ataxic, the right being the same as the left, but the arms slightly more than the legs. The voluntary movements of the hands were so ataxic that she could scarcely hold and drink a glass of water. The incoordination in the lower extremities made standing and walking quite difficult. There was no Romberg sign. All the incoordinations were typically cerebellar in type. Cerebellar asynergia was evident. Two days later not much change was to be noted. Ten days later a great improvement was found. Seven weeks after the onset of the neurologic sequel, almost no trace of the clinical signs of involvement of the nervous system was present.

I believe that in the case just cited we have had a pathologic process involving esesentially the cerebellum. Possibly other portions of the encephalon were involved in a very minor degree. A complete recovery in a short period indicates that few or no cerebellar neuronal elements were destroyed. There was present an inflammatory process, either the result of an intoxication or directly from some secondary organism or from the hypothetic virus of measles. The termination precluded a necropsy. A spinal puncture was denied.

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The only parallel case has been found after searching the literature. A. W. Fairbanks,[7] under the title "Ataxia Following Measles," cites a case indicating an involvement of the cerebellum during convalescence following measles. However, there were evidences of added involvement of the right motor neural tracts. E. Apert [8] mentions a form "rougeole ataxo-adynamique."

CASE 2.—*A meningitis complication.—History.*—L. H., a girl, aged 4 years, whose family and past history was negative, had been a healthy, well developed child. In July, 1919, I consulted with Dr. Frank Neff, who had taken charge of the case a few days previously, and about ten weeks after the attack of measles. There was present with the measles a bronchopneumonia. An otitis media followed. The child did not leave the bed after the measles and the subsequent complications. There was a state of marked helplessness, inability for any coordinated movements, and the child was inarticulate. Dr. Neff in his examination found a negative von Pirquet reaction; a white cell count of 5,400; a negative Wassermann reaction on blood and spinal fluid, and a spinal fluid clear, with no pleocytosis or bacteria.

Examination.—The patient was seen in bed, entirely helpless and speechless. She recognized no one. There was present much psychomotor unrest, with constant purposeless movements of hands and feet. There were no true palsies. There was some response to spinal stimuli. The superficial reflexes were present. The deep reflexes were present in the upper extremities, brisk at the right patellar and Achilles tendons, and feeble at the left. The Babinski and Oppenheim reflexes were questionable. The pupils responded to light and accommodation, but to a diminished degree. The eye grounds were nearly normal. Vision and hearing were undoubtedly present.

A neurologic examination in September indicated much improvement. Walking was possible.

A neurologic examination eight months after the first one indicated much favorable progress. The patient walked and played some and recognized her parents. She was unable to talk. Movements were not perfectly coordinated. Petit mal convulsions had been present during certain periods.

This patient undoubtedly had a meningitis with the other complications following the measles. The type of secondary meningeal infection was never determined. A cerebritis with a particular involvement of

7. Fairbanks, A. W.: Arch. Pediat. **24**: 770 (Oct.) 1907.
8. Apert, E.: Traité de médecine et de thérapeutique, Paris, **2**, Eruptive Fevers, p. 201, 1915.

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the cortex, as so frequently happens with meningitis, was evident. In the way of prognosis, it is reasonably certain that imbecility and possibly epilepsy will always be present.

THREE GROUPS OF CASES

In a concluding examination of the subject of brain complications and sequelae appearing in the course of or following measles, after a critical review of the reported cases and discussions in the literature and an analysis of my own few cases, I would make three groups:

1. The first group would include a certain minor number of cases in which the relationship of the measles would be merely incidental. Some of the cited cases have the onset of the brain troubles as first occurring several weeks to a few months following the eruption. Naturally the bearing of the measles infection to such sequelae can be questioned with justice. Of course, bacteria of various kinds may be harbored for a long period and under certain conditions be released as a local or general infection, when resistance in the host has been lowered by a disease such as measles.

2. The second group would include the secondary infections due to various organisms. The bacteria may be delivered to the central nervous system by the blood stream. However, the mode of invasion is undoubtedly in most instances through the cribriform plate from the nasopharyngeal cavities. This route of infection for *Diplococcus intracellularis-meningitidis* and the acute poliomyelitis virus is now well recognized. A great many more cases of meningitis follow measles, compared with all the other complicating disorders in the central nervous system. The fact that a large majority of the complications have their onset a few days following the disappearance of the eruption or during the early convalescence speaks for the importance of this second group. The interval is in accordance with the incubation period required for most of the organisms for secondary infections.

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3. The third small group is a less certain one, owing to the fact that the exact etiology for measles is as yet undetermined. Clinical studies of the disease lead us to believe that the blood stream is teeming with the virus. Accordingly, the vascular channels of the meninges and brain may readily become involved. Certainly, some of the brain complications first manifested during the eruptive stage or the early convalescent period may be caused directly by the unknown virus of measles. Even some of the troubles appearing later in the convalescence may be due to this cause.

The prognosis for these brain disorders certainly depends on the brain tissue changes. If there has been much involvement of the leptomeninges or destruction of many important neurons, complete recovery is uncommon. From reports of good recoveries within from one to four months, I infer that the pathogenesis was mild. It was mild in my case with ataxia, which made a complete recovery in a few weeks; and severe in the meningitis case, in which entire recovery could hardly be anticipated.

The management and treatment of these cases depends on the lesions and the attendant deformities or deficiencies. Prophylaxis is first in importance. No unnecessary exposures to measles should be tolerated. Then the disease should be given careful attention, particularly as to preventing complications. Possibly many of the complications with meningitis reported might have been avoided had some attention been given the upper respiratory passages.

CONCLUSIONS

Cases of measles are sufficiently important to warrant careful consideration. Involvements of the brain in the course of measles, while apparently not as frequent as in other infectious diseases, are occasionally encountered. It is possible that many have not been diagnosed or recorded.

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I have seen in my consulting practice and in the clinics of other neurologic consultants, certain mild neurologic or mental cases that were difficult to catalogue. This includes some with the neurasthenic syndrome. They may have originated from troubles in the brain or meninges in the course of or following measles and other exanthems. We can trace some of these neurologic disorders back to early childhood without knowing the exact etiology. I believe that at least a portion of these can be attributed to permanent pathologic residues in the encephalon or its leptomeninges, directly caused by the toxin or virus of measles or from one of the complicating secondary infections.

ABSTRACT OF DISCUSSION

Dr. Karl A. Menninger, Topeka, Kan.: In the consideration of mental defect or feeblemindedness we meet those who hold to the hypothesis, not substantiated, that cases of feeblemindedness are "born and not made." We should not be too hasty in drawing a distinction between mental and physical forces even in feeblemindedness. Until we know what feeblemindedness is, it behooves us not to be too arbitrary in regard to conditions of feeblemindedness as being "born and not made." Kraepelin put forth the doctrine that there is a qualitative specificity in the mental symptoms and psychiatric product of the infectious diseases. That is, if keen enough in clinical work, we should be able to differentiate the physical disease from the mental symptoms alone. Our influenza studies have tended to show that possibly instead of a qualitative specificity, it is a quantitative specificity that holds. This is simply to say that one disease evokes many neuropsychiatric sequels. Dr. Skoog's report concerning measles might be interpreted as further confirmation of this view. Measles is quite comparable with influenza in morbidity rates; and of many cases of influenza a comparatively large number showed nervous system complications, whereas of the equally numerous cases of measles each year, apparently few exhibit nervous system pathology. The neurotoxic index of influenza is high; that of measles is low; the difference is quantitative, not qualitative.

Dr. A. L. Skoog, Kansas City, Mo.: We should be on our guard constantly when making neurologic examinations, especially in cases in which the etiology is obscure and uncertain, to determine whether the actual cause of the disease may not rest in some long-since forgotten antecedent disease such as measles.

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THE ETIOLOGIC RELATION OF WORMS TO EPILEPSY

E. BATES BLOCK, M.D.
ATLANTA, GA.

Although the idea that the animal parasites of man are among the causes of epilepsy is generally known, there has not been any definite proof or disproof of this theory, and no adequate explanation of the method by which they provoke convulsions.

Some of the leading textbooks do not mention worms as a cause of epilepsy, while others mention them only with considerable reservation. To quote some of our leading writers, Oppenheim [1] only mentions worms as among the diseases to which epilepsy has been attributed, and even in discussing infantile eclampsia [2] he says the causal effect of intestinal worms, which is supported by earlier experience and by recent observations (Naab, Festa), has been disputed by many writers, such as d'Espine and Bong.[3]

Gaupp [4] mentions worms as being "a more or less justifiably regarded . . . exciting cause," but regards it as a "secondary determining cause."

Osler [5] mentions worms under reflex causes among other subjects "given as causes."

Dana [6] rather significantly says more cases occur in the country than in the city, more in temperate climates. "The so-called reflex causes are . . . worms, etc."

Gordon [7] says: "The relation of epilepsy to . . . intestinal worms is maintained by some observers, but not definitely proven."

1. Oppenheim: Textbook of Nervous Diseases, 1911, p. 1203.
2. Oppenheim: Textbook of Nervous Diseases, p. 1233.
3. D'Espine and Bong: Ann. de méd. et chir. infant. 1902.
4. Gaupp, in Curschmann's Textbook on Nervous Diseases, 1915.
5. Osler: Principles and Practice of medicine, 1917.
6. Dana: Textbook of Nervous Diseases, 1915.
7. Gordon: Diseases of the Nervous System, 1913, p. 491.

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Gowers[8] says: "The chief peripheral irritation which can be regarded as a cause is that of intestinal worms."

Bing[9] says: "The convulsions of children having intestinal parasites do not usually appear to be of epileptic character." Later, he says: "Epilepsy is no neurosis," and "Hippocrates has rightly remarked that its etiology and pathology are in the brain."

Church and Peterson[10] say: "Reflex irritations due to . . . intestinal worms . . . are occasionally sufficient to provoke epileptic attacks and to cause their repetition."

Among Spratling's[11] cases there were only two cases attributed to worms, and Gowers[12] reported only six. The latter says that after the convulsions have lasted for some time the epilepsy is not cured by getting rid of the worms, It should be borne in mind that these authors wrote at a time when no systematic examination of the stools were made, and the importance of close investigation of the subject was not so apparent as it is at the present time.

Two questions are considered especially in this paper, namely:

1. Are we justified in regarding worms as one of the causes of epilepsy?

2. If worms cause epilepsy, how do they do it?

1. (*a*) In a paper read before the Southern Medical Association in November, 1919, I[13] reported 200 unselected cases of epilepsy in my private records. No systematic examinations of the stools were made, and the subject was not made a special object of research, but in thirty-seven cases, worms or the eggs of worms were found in the stools. Of these, two were *Taenia;* thirteen, *Ascaris;* seventeen, hookworm, and three, *Oxyuris*. In one case, *Taenia saginata* and hookworm

8. Gowers, in Allbutt's System of Medicine, 1900, p. 768.
9. Bing: A Textbook of Nervous Diseases, 1915, p. 396.
10. Church and Peterson: Nervous and Mental Diseases, 1914, p. 641.
11. Spratling: Epilepsy, 1904.
12. Gowers: Epilepsy, 1885.
13. Block: The Etiology of Epilepsy, J. South. M. A., 1920.

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were found together in repeated examinations. In three cases no record was made as to the kind of worms.

Since then I have compiled records from my private cases of epilepsy, taking them alphabetically, but only the cases in which there was a definite report made as to the presence or former presence or absence of worms or eggs in the stools, with the following results: Out of 100 cases of epilepsy in which the stools were examined, fifty-six were negative; twenty-one showed *Ascaris;* seventeen showed hookworm; four showed *Oxyuris;* one *Taenia* (*Hymenolypis*) *nana;* one *Taenia saginata. This gives a total of* 44 *per cent. of the cases of epilepsy having worms.* In seventeen of the cases the worms or eggs were present on examination at the time of consultation, while in twenty-seven cases they had been previously found in laboratory examinations or had been seen by the patients and accurately described. Most of the cases of *Ascaris* belonged to this latter group.

(*b*) The work done by the Rockefeller Sanitary Commission under the direction of Dr. A. G. Fort [14] showed that out of a total of 35,133 examinations, 11,418 people showed intestinal parasites (32.49 per cent.). As part of these were double infections, a total of 11,985 worms was found, which are classified in Table 1. The apparent discrepancy in figures is due to double infection.

(*c*) Gage and Bass [15] examined ninety students in the Tulane University and found 42.4 per cent. of those, from the country, and 2.5 per cent. from the city infected with hookworm, or a total of 20.7 per cent. of young male adults. Of the mill population, they found that 12.6 per cent. have hookworm.

(*d*) Out of the last 1,000 cases examined in the laboratory of the Georgia State Board of Health,

14. Fort: Report of the Rockefeller Sanitary Commission for the Relief and Control of Uncinariasis, p. 24, 1910-1915. The discrepancy in percentages was a verbal communication.
15. Dock and Bass: Hookworm Disease, 1910, pp. 39, 41.

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under the direction of Dr. T. F. Abercrombie, 250 showed worms (25 per cent.). Out of 1,695 stools examined (some of which were reexaminations of the same patient), 680 stools showed worms (40.12 per cent.). Naturally, most of the stools sent to the state board of health are sent because worms are suspected; and if we accept their experience of 25 per cent., we must readily admit that it is far in excess of the number infected in Georgia. The kinds of worms found are given in Table 1.

TABLE 1.—KINDS OF WORMS FOUND

	No.	Per Cent.
Out of 44 cases of worms with epilepsy in my practice:		
Ascaris	21	47.17
Necator americanus	17	38.63
Oxyuris	4	9.09
Taenia nana	1	2.27
Taenia saginata	1	2.27
Rockefeller Commission, 11,985 kinds of worms:		
Ascaris	1,240	10.34
Necator americanus	10,186	85.00
Oxyuris	11	.09
Taenia nana	502	4.19
Taenia saginata	5	.04
Strongyloides	29	.24
Taenia solium	2	.02
Trichocephalus	10	.08
Georgia State Board of Health, 680 kinds of worms:		
Ascaris	22	3.23
Necator americanus	596	87.66
Oxyuris	0	
Taenia nana	51	7.50
Taenia saginata	3	.44
Trichocephalus dispar	2	.29
Double infection: hookworm and Taenia nana	6	.88

(*e*) Certainly we cannot accept the results of a series of examinations in *suspected* cases as an index of the number of people in a community who are infected by worms, because the vast majority of people are well, worms are not suspected, and therefore no examination is made. We come to the following comparisons: that 44 per cent. of my cases of epilepsy were *known* to have worms, while 20.7 per cent. of the Tulane University students had worms without any probability that many of them had epilepsy. Their observations that only 2.5 per cent. of city students had worms is very significant when considered in conjunction with my observations that the vast majority of my cases of epilepsy lived in the country or small

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towns, which is also supported by Dana's experience. Ammann [16] states there are twice as many in the country as in the cities in Switzerland.

We have, then: the Rockefeller commission, 32 per cent.; Gage and Bass, 20.7 per cent.; Georgia State Board of Health, 25 per cent.; in epilepsy, 44 per cent.

(*f*) *The question naturally arises, How many people have epilepsy?*

Spratling found: in Switzerland, 1 to 750 (Kolle); in Russia, 1 to 2,000 (Kovalevsky); in Russia, 1 to 1,000 (Shoutelwort); in Scotland, 1 to 750 (Pelmoun); in France, 1 to 1,100 (Vernet); in the United States, 1 to 500 (Peterson). The highest figures known to me are those given by Ammann,[16] namely, 0.5 per cent. of the population of Switzerland are epileptics (20,000 people). In my private cases I find 64 per 1,000 have epilepsy, or 6.4 per cent., which is of course high on account of the character of my practice being almost exclusively neurologic. Nevertheless, even these high figures show that the chances of a fortuitous occurrence of a high percentage of worms with epilepsy is greatly out of proportion to the ordinary expectancy.

TABLE 2.—KINDS OF WORMS FOUND IN PATIENTS WITH EPILEPSY AND WITHOUT EPILEPSY

	Without Epilepsy	With Epilepsy
Hookworm	172	8
Ascaris lumbricoides	6	0
Taenia nana	9	0
Trichocephalus	1	0
Oxyuris	1	0
Taenia nana and hookworm	0	1
Taenia nana and ascaris	0	1
	189	10

Total cases, 199.

(*g*) *The question arises also, If 44 per cent. of the cases of epilepsy have worms, then how many cases of worms have convulsions or epilepsy?* To determine this point, letters were sent to the physicians of Georgia who had sent stools to the state board of health, and I

16. Ammann: Die Erkrankung und Sterblichkeit an Epilepsie in der Schweiz, Inaug. Diss. Zurich, 1912; Neurol. Centralbl. 32: 575, 1913.

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wish here to thank them for their cooperation in obtaining these statistics, and also Dr. T. F. Abercrombie and Dr. T. F. Sellers for allowing me to utilize their work. Out of 199 replies: Ten cases of worms had convulsions or epilepsy; 189 cases of worms did not have epilepsy. As to the kind of worms, I present Table 2. It will therefore be noted that in two cases of worms associated with epilepsy there was double infection, and it is therefore difficult in these cases to say which may have caused the epilepsy.

It is obvious from this paragraph that only 5 per cent. of the cases of intestinal parasites had convulsions or epilepsy, and I shall later consider the question as to whether their mere presence in the intestinal canal is sufficient to produce epilepsy.

(*h*) *From these statistics we can say that worms are more frequent in association with epilepsy than we find them in the examinations of people who have not, as far as we know, epilepsy, and I believe we are justified in accepting it as one of the causes of epilepsy.*

2. Having accepted worms as one of the causes of epilepsy, the next question is, How do they do it?

We will not take time to discuss Aristotle's "if the brain is willing," or Féré's "spasmophilia," or Joffroy's "convulsive aptitude." These, I think, are generally conceded.

(*i*) The general ill health of the patient would, of course, render the nervous stability and equilibrium of the patient less.

(*j*) The reflex irritation may act as a factor in a summation of stimuli.

(*k*) Peiper has advanced the theory that intestinal parasites produce toxins.

(*l*) The possibility of the actual invasion of the brain by the larvae of the worms.

(*i*) When worms are present the *general health* of the patient suffers, and various well known symptoms occur. The majority of cases show, however, no evi-

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dence of ill health and a comparatively small number show anemia. Out of *twenty-three* of my 100 cases of epilepsy with worms in which the blood was examined, *seven* showed a reduction of hemoglobin below 80, while only four showed a reduction of red blood corpuscles below 4,000,000.

TABLE 3.—BLOOD COUNT IN PATIENTS WITH WORMS

Hemoglobin	Red Blood Cells	Leukocytes	Kind of Worm
70	4,592,000	5,200	Taenia nana
80	4,816,000	7,600	Ascaris lumbricoides
90	4,110,000	6,800	Hookworm
78	4,730,000	6,600	Ascaris
85	4,830,000	6,450	Ascaris
75	3,890,000	5,200	Ascaris
75	5,580,000	7,400	Hookworm
75	4,640,000	8,400	Ascaris
90	5,940,000	10,600	Hookworm
80	3,970,000	3,800	Hookworm
85	4,988,000	5,100	Ascaris
85	49,880,000	6,200	Pinworm
85	4,810,000	6,600	Hookworm
90	4,200,000	4,000	Pin worm
80	4,210,000	6,600	Hookworm
85	4,148,000	7,000	Ascaris
80	4,536,000	4,900	Ascaris
85	4,200,000	5,600	Hookworm
80	3,700,000	4,000	Ascaris
60	3,700 000	8,200	Ascaris
80	5,140,000	10,000	Hookworm
80	4,530,000	6,150	Hookworm
70	4,030,000	6,200	Ascaris.

We know, however, from experience that general anemia is not a cause of epilepsy, while the theory of a local cerebral anemia as an immediate cause of the onset of the convulsions has been well argued by Russell.[17] General ill health from other causes has not been invoked to explain epilepsy, and there is little to support this theory, especially as the general health of epileptics is often good between spells.

(*j*) The theory of a *reflex irritation* is somewhat vague, but we are all familiar with the fact that children sometimes have worms and convulsions and the latter disappear on geting rid of the worms, although as a rule this is, unfortunately, not true. We know that people who have a tendency to convulsions will often develop them from a slight additional cause, such as overeating, eating certain foods which are difficult

17. Russell, A. E.: Some Disorders of the Cerebral Circulation and Their Clinical Manifestations, Lancet 1: 962, 1031, 1093, 1909.

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to digest, constipation, etc. Only one out of eight of the cases of convulsions of infancy go on to the protracted condition of repeated attacks which we term "epilepsy."

(*k*) Peiper [18] regards the nervous symptoms from *Ascaris* infection not as a reflex but rather as due to a *toxin* contained in the helminths, or metabolic in origin. "With regard to the toxic action of oxyuris there is only the single record of Hartmann, who noticed the disappearance of epileptic fits and psychic disturbances in a girl, aged 13, after the removal of oxyuris" (Fantham). A general discussion of the evidences of toxic action of cestodes and nematodes will be found in Fantham's [19] book (p. 644 et seq.). The main contention of those who support this theory is that the poisons elaborated by the parasites produce anemia.

(*l*) As to the possibility of the actual *invasion of the brain by the larvae* of worms, this finds its greatest support in those cases in which cyst formation is prominent, and leads to easy detection of the lesion on account of its size and often the great number, as in the case of Egerton Brown,[20] in which they occurred in all parts of the brain as well as other organs of the body. No signs of adult worms were found either during life or in the intestines at necropsy. A similar case was reported by Vigouroux and Hérisson-Laparu [21] in which, as in the former case, there were numerous epileptiform fits with numerous cysticerci in the brain. According to Fantham, in "cysticercus of the brain ten to nineteen years may elapse from the first appearance of cerebral symptoms until death" (pp. 336-337). He quotes also the postmortem records of the pathologic institute in Berlin before the year 1875, which showed 20 per cent. cysticerci affecting

18. Peiper: See Fantham, p. 649. Vide Seifert: Lehrb. d. Kinderkrankh., 1897, p. 243.
19. Fantham, Stephens and Theobald: The Animal Parasites of Man, 1916.
20. Brown, Egerton: A Case of Cysticercus Cellulosae Occurring in the Insane, Rev. Neurol. & Psychiat. 1906, p. 272.
21. Vigouroux and Hérisson-Laparu: Soc. anat. de Paris, March 7, 1913; Presse méd., No. 23, March 19, 1913, p. 225.

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the brain, while of late years it has fallen to 1 per cent. (Orth [22]), and in the clinic of de Amicis at Naples among seven cases of cysticerci of the skin they were found four times also in the brain (Sipari [23]). In 155 cases of cysticerci compiled by Stiles, the parasite in 117 was found in the brain (Osler, p. 288).

While the invasion of the whole body and brain by *Cysticercus cellulosae* (*T. solium*) is very common in Europe this parasite is rarely found in America. A case is reported by Lloyd [24] in which the symptoms were those of brain tumor without epileptic attacks, but no tapeworm was found in the intestines. In other cases the epilepsy is of a jacksonian type such as that reported by Fischer.[25] In Paechell's [26] case the jacksonian epilepsy was on one side of the body and later on the other side. There were about seventy cysts, but no tapeworm was found. An equally large number of cysts with jacksonian epilepsy was found by Ziveri.[27] In Saenger's [28] case of epilepsy there was no necropsy, but cysts were found under the skin. Other cases with epileptiform attacks or convulsions were reported by Goldstein [29] and Biondi.[30]

References to forty-four articles on cysticercus in the brain are contained in the Index to the Surgeon-General's library.

The pathologic changes in the brain found by Krause [31] seem quite sufficient to form a basis for the production of epileptic fits.

That other forms of taenia invade the brain is shown by the presence of *Cysticercus acanthotrias* Weinland

22. Orth: Berl. med. Ges., June 29, 1904.
23. Sipari, Angelo: Trani. Neapel., 1900.
24. Lloyd: A Case of Cysticercus Cellulosae of the Brain, Philadelphia M. J., March 19, 1898, p. 516.
25. Fischer: Ueber Folgezustände kleinster Läsionen in Bereiche des motorischen Armzentrums nebst einem Beitrag zu cysticercosis cerebri, Monatschr. f. Psychiat. u. Neurol. **18**:, No. 2.
26. Paechell: A Case of "Status Epilepticus" and Death Due to Cerebral Cysts of Cysticercus Cellulosae, J. Ment. Sc., 1916, p. 180.
27. Ziveri: Un caso di cisticerco del cervello, Riforma med., 1909.
28. Saenger: Zystizerkenepilepsie, Neurol. Centralbl., 1913, p. 729.
29. Goldstein: Ein Beitrag zur Lehre der Zystizerkerkose des Gehirns und Rückenmarks inbesondere der Meningitis cysticercosa, Arch. f. Psych. u. Nervenkr. **49**.
30. Biondi: Contributo allo studia della cisticercosi cerebrale, Riv. Ital. di. Neuropatol. Psichiat. ed. Elettroter. **10**: 10-21, 51-69, 1917.
31. Krause: Monatschr. f. Psych. u. Neurol. **31**, 1912, ref. Neurol. centralbl., 1913, p. 373.

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in the dura mater, which, however, Fantham regards as a form of *C. cellulosae.* In discussing somatic taeniasis, Osler states that *Cysticercus taeniae-saginatae* has been found only two or three times in man (p. 287). According to Fantham (p. 341), *Taenia saginata* gives rise frequently to cysticerci which invade the muscles of the ox, especially the pterygoid muscles, and has been reported in the human brain by Arndt,[32] and Nabiers and Dubreith.[33] As to *Taenia hymenolepis,* no mention is made by Fantham of their presence in the brain, but he mentions their causing epilepsy (p. 324), and in the first case reported (Bilharz, 1851) the boy died of "meningitis." On p. 649 he says: "How far the serious disturbances of the nervous system frequently to be observed in *Hymenolepis nana* are to be considered as of purely reflex nature, or toxic, must remain an open question: the same applies to *Dipylidium caninum.*"

As *Taenia crassicollis* (cat) and *Taenia serrata* (dog) habitually pass from the intestines into the portal vein and leave the liver with the circulation and go to various organs of the body, it would be easy to see how they may also pass to the brain, though I have no direct report on this fact.

As to *Taenia echinococcus,* we frequently saw hydatid cysts in the brain, often calcified, in the necropsy room in Prague. Osler (p. 291) states that out of 1,634 collected cases of hydatid cysts, they occurred in the nervous system in 122, and he quotes Davies Thomas of Australia, who tabulated ninety-seven cases of echinococcus cyst, including some of *Cysticercus cellulosae,* and found them most frequent in the cerebrum. Other cases have been reported by Foresti and Bonaba [34] and Eichhorst.[35] In the cases of Maunsell [36]

32. Arndt: Ztschr. f. Psychiat., **24.**
33. Nabiers and Dubreith: J. méd. Bordeaux, 1889-1890, p. 209.
34. Foresti and Bonaba: Bull. et mém. Soc. méd. d. hôp. de Paris **38:** 262, 1914.
35. Eichhorst: Ueber multilokularen Gehirnechinokokk, Deutsch. Arch. f. klin. Med. **106,** 1912.
36. Maunsell: Subtentorial Hydatid Tumor Removed by Trephining, New Zealand M. J. **11:** 151, 1888-1889.

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and Newton[37] the symptoms were those of brain tumor, with convulsions several times a day in the former. The meninges of the spinal cord are sometimes affected, as in Marwood's[38] case. All of which go to show the not infrequent invasion of the nervous system by *T. echinococcus*. References to thirty-nine articles on echinococcus in the brain may be found in the Index to the Surgeon-General's library.

The mode of production of epilepsy by *Ascaris lumbricoides* is a more difficult problem. There is considerable evidence that Peiper's assertion was right, that the ascaris produces a poison which has a profound effect on the nervous system. Serious motor disturbances and fequently death were produced by Messineo[39] by injecting extracts of the worms, and meningitic symptoms may be produced (Fantham, p. 649). As to the invasion of other organs, ascarides, oxyuris and trichocephalus have frequently been reported to penetrate the intestinal wall. I have in my collection a specimen of necrosis of the liver due to an ascaris ascending the bile ducts, and females have been reported (Fantham, p. 689) to lay eggs in the liver. Just what becomes of these eggs, and what becomes of the ascarides which become free in the peritoneal cavity is not known to me. Ascaris ova have been found encapsulated in the peritoneum, and they have been known to produce nodular or abscess formations. In the case of oxyuris they form small nodules in the peritoneum, which shows their power of penetration of other tissues. Symptoms of meningitis may be produced by *Trichocephalus dispar,* but whether this means an invasion of the meninges by the larvae or merely a toxic action has not yet been determined. The case of Lutz[40] in which two ascarides were found in

37. Newton: A Case of Hydatid Cyst in the Cerebellum, Australasian M. Gaz. **22**: 219, 1903.
38. Marwood: Paraplegia: The Result of Hydatid Within the Membranes of the Spinal Cord, New Zealand M. J. **11**: 137, 1888-1889.
39. Messineo: Goirn. med. del regio escc., 1905.
40. Lutz: Wien. klin. Wchnschr. **15**, 1905 (quoted by Fantham).

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the pulmonary artery was due to a gunshot wound of the abdomen. Of course their occurrence in the frontal sinus, in the antrum of Highmore, the lacrimal duct, and other narrow passages, has a different explanation but shows their migratory tendency. Whether their ova ever find their way to the brain and set up a focal irritation there remains an open question. The case of St. Deák [41] in which the epilepsy and pyromania were cured by santonin, while not an isolated experience, is not the rule.

Fantham (p. 639) quotes Inouyés' report of nineteen cases of paragonimiasis of the brain with general convulsions on eight occasions, unilateral convulsions on six occasions, convulsions with paralysis on the same side and hemiplegia five times each, and they quote several other authors who found the paragonimus in the brain with jacksonian epilepsy. According to them, paragonimiasis of the brain appears to arise by embolism from a primary pulmonary lesion (lung flukes).

The well known tendency of the hookworm (*Ancylostoma duodenale* and *Necator americanus*) to invade most of the organs of the body leaves little if any doubt that they also reach the brain, although I have seen no definite reports on their being found there. Looss [42] has shown experimentally that the larvae enter the skin and are found in the veins, arteries and lymphatics, through which they pass to the heart and lungs, and there is no reason why they should not reach the brain. He says: "I cannot help thinking that in those cases, by no means rare, in which the anemia in man will not yield even after the adult worms have been completely or almost completely purged, the chief cause of this obstinacy might be found in strayed larvae still present in the body." Stiles has demonstrated the hookworm in most of the

41. St. Deák: Ein Fall von Pyromanie, bedingt durch ascaris lumbricoides, Orvosi Hetilap, 1911; abstr. Neurol. Centralbl., 1912, p. 706.

42. Looss: Records of the Egyptian Government School of Medicine, 4, 1911.

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organs of the body. Fantham suggests that certain observations would tend to show that ancylostome anemia may be due to toxins excreted by the parasites.

We know also that other parasites invade the body tissues. Osler states that trichinae pass through the intestines into the venous system and by the blood stream into the muscles. Bunzl[48] found numerous nematodes in the brain of a common mole resembling the embryos of *Trichina spiralis.*

CONCLUSION

From the foregoing observations I feel that we are justified in stating that *there is reason to believe that epilepsy when produced by animal parasites is due to the actual invasion of the brain by the larvae.* The theory is at least less vague than the other theories, and is supported by the clinical fact that the epilepsy is rarely cured by freeing the intestinal tract of the parasites.

The absence of worms or eggs in the stools is not proof of the absence of parasites in other parts of the body.

ABSTRACT OF DISCUSSION

Dr. Julius Grinker, Chicago: Have you had an opportunity to perform a necropsy on any of your cases, and, if so, have you been able to demonstrate to your satisfaction the presence of worms in the brain?

Dr. David S. Booth, St. Louis: I do not suppose there is any doubt that worms do cause epileptic attacks under certain conditions. The question is, how often? I have found it very infrequently. Professor French of the Southern Illinois Normal University discovered a new worm in a few cases of epilepsy, and on ridding the body of them, the patient recovered from the attacks. Professor French named that worm, or parasite, *Gastrophilus epilepsalis.* He made a great many observations of stools and in more than one thousand cases he found less than a dozen persons infested with this worm.

The method by which these parasites cause attacks probably is through damage to the intestinal canal. In one case seen by me, no focal cause of the trouble could be found, but a proctologist discovered a rectal ulcer, the healing of which resulted in absolute cure of the attacks. The patient has

43. Bunzl: Arb. a. d. Neurol. Institut Wien **11**, 1904.

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remained cured now for eight or ten years. So that, in certain cases, at least, there must be something in the condition of the bowel. In many cases we find that the tongue is particularly foul and especially before or immediately after the attack, indicating, if we accept the usual interpretation of this condition, the presence of autointoxication.

Dr. H. Climenko, New York: I have observed a case of *Cysticercus* which was diagnosed during life and examined postmortem. In that particular case there were no convulsions. It is readily conceivable that worms will produce convulsions, causing them either on an organic basis or on a basis of toxemia. I cannot, however, conceive that idiopathic epilepsy, which usually occurs at the time of puberty and goes on through life at periodic times, could be caused by worms. It appears to me that if the worms are found, they are simply a contributory cause, such as any other cause could be, either of psychogenic or of metabolic origin. Idiopathic epilepsy is due to some other causes not yet known. The latest theory, the impairment of the elements of metabolism due to disturbance of the internal secretions, seems to be much more tenable than that of worms. Were worms responsible for epilepsy, pathologic reports of cases of epilepsy would more frequently mention changes in the brain or in the meninges than have been mentioned hitherto.

Dr. Marvin L. Graves, Galveston, Texas: Discussion of this subject hinges on what epilepsy is. A well known textbook on nervous diseases gives this definition: Epilepsy is a progressive paroxysmal psychoneurosis. Evidently the author intended to limit the occurrence of convulsions to the old designation of idiopathic epilepsy. Terms such as epileptiform and epileptoid, emphasize certain convulsions associated with toxemia and have a close resemblance to so-called "idiopathic" epilepsy. These may be caused by both animal and vegetable parasites. I have seen epileptiform convulsions similar to "idiopathic" epileptic convulsions, caused by the typhoid bacillus, and convulsions due to malarial parasites which have been entirely relieved by the administration of quinin. One year ago I saw a child suffering from frequent epileptiform attacks, associated with the classical symptom complex of spru. Large numbers of the organisms were present in the stools and also in the cultures from the dorsum of the tongue, and Crombre's ulcers were present. The morphology and cultural characteristics of the fungus corresponded to the *Monilia psilosis* of Ashford. An autogenous vaccines was made and given and the child was dieted and these organisms disappeared from the stools, but the epileptiform attacks continued. I have seen a considerable number of cases of epileptiform attacks associated with hookworm and other intestinal parasites, and yet the epileptic symptoms continued after the administration of drugs which destroy the hookworm. So I believe it has not yet been

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definitely ascertained that any of these intestinal parasites are productive of the disease that we have always recognized as "idiopathic epilepsy." I am in entire accord with Dr. Block that we do not know what idiopathic epilepsy is, and yet, simply because we have not discovered a cause for it, this does not mean that these attacks do not have some definite origin or etiologic factor.

Dr. Tom A. Williams, Washington, D. C.: The etiologic factors of the epilepsies are becoming more and more numerous. We should not fail to speak of the clinical findings of the older physicians, who believed that worms were a cause of convulsions. Is that clinical knowledge well founded or not? Dr. Block's solution is not satisfactory. It is subject to certain criticisms and exceptions and demands investigation. Not all lesions produce epileptiform attacks, even such gross lesions as tumor, syphilitic encephalitis, acute encephalitis, frequently fail to produce attacks of epilepsy, and that failure does not seem to depend on the site of the lesion. There must be another factor. We find epilepsies produced in such functional disturbances as uremia, more particularly the uremic state seen during parturition, which epileptic attack disappears after the cause is removed. We can produce epileptiform attacks by the injection of absinthe. May not all epilepsies be caused by a metabolic factor similar to toxemia? These patients apparently are well between attacks. So although there may be a gross lesion in the brain which produces the attack, I have inferred that there is also metabolic cause. While worms cause epileptiform attacks, the condition might not be due to the presence of worms in the brain, as suggested by Dr. Block, but to a metabolic cause made worse by the presence of the worms.

Dr. E. Bates Block, Atlanta, Ga.: I believe I demonstrated that worms may cause epilepsy, and that the incidence of worms in epilepsy is far greater and more frequent than we had supposed. We cannot ignore them as a causative factor. As none of my patients died, a postmortem examination could not be made. It would be quite a task to find a larva in the brain with the microscope. If the hookworm gets into the brain one of two things must happen: Either the worm dies and is absorbed, or, if the worm remains there long enough without dying and being absorbed, there will ensue a reactive protective pathologic effect whereby tissue organization will take place with the formation of a scar. There may be many of these scars in the brain and they may become the focus of irritation. Following an operation on the brain, which causes the development of scar tissue, epilepsy rarely occurs, and yet trauma to the brain is a cause of epilepsy. The vast majority of syphilitics do not develop epilepsy, and yet syphilis is an occasional cause of epilepsy.

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ENCEPHALITIS LETHARGICA

EDWARD LIVINGSTON HUNT, M.D.
NEW YORK

In the late summer of 1918 there appeared in New York several cases of what seemed to be a new disease. These cases were characterized by temperature, apathy and various cranial nerve paralyses. By the early fall the number had increased and so continued during the winter. As summer approached, the disease abated, but reappeared in the fall of 1919, and this time with greater virulence than ever. The condition was recognized as being encephalitis lethargica.

Study of medical literature brought to light the fact that in 1890 a similar series of cases had originated in Hungary and later had spread to Germany, Austria and other parts of Europe, and that several years later a second and more extensive outbreak had occurred involving almost all of central Europe.

There were the slow cases, the rapid and the mild. The slow form of encephalitis lethargica begins gradually and insidiously. There are the premonitory symptoms of malaise, coryza, headache, dizziness, slight temperature and a tendency to sleep. These symptoms slowly grow worse, and at the end of a week are augmented by double vision or other cranial nerve paralyses, gradually rising temperature, a marked degree of apathy, and somnolence. From this time on innumerable symptoms may be added, all, however, indicating an involvement of the central nervous system and in the rare instances of the peripheral nervous system. Mental symptoms are often superadded. These vary greatly and may be maniacal, depressive, confusional, catatonic, or all combined. In this form basilar symptoms

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are the most constant and are almost uniformly present, showing that the principal seat of the disease is in the region of the medulla and pons. Spinal cord symptoms may appear later in the form of rigidity, pain from root involvement, and loss of sphincter control. There is usually some tremor and chorea.

The rapid form of encephalitis begins suddenly with a marked rise of temperature to 102 or 103 and several well-pronounced symptoms. These are delirium, mania, pain, double vision, twitching and cranial nerve paralyses and marked apathy. In this form the course is shorter, the prognosis more grave and the tendency to sphincter involvement greater. In both of these types I found several common symptoms. These were: (1) hypersomnia; (2) ocular nerve paralyses; (3) temperature, and (4) a change in the general state. It is, therefore, fair to call these four symptoms "the cardinal symptoms" of encephalitis.

These so-called cardinal symptoms deserve a few moments' consideration.

1. *Hypersomnia.*—This appears early in the rapid cases, and at the end of a few days in the slow types. It differs from any other form of stupor. The patient can be roused, and when roused appears quite himself; but in the midst of the conversation he is very apt suddenly and without warning to drop back into a profound sleep. No stertorous breathing accompanies it; rather does the patient appear to be sleeping quietly and profoundly.

2. *Ocular Nerve Paralyses.*—These are of several kinds. There may be double vision, ptosis which is unilateral or bilateral, nystagmus, strabismus or external rectus paralysis. The most common of all is the diplopia; in many instances it is the earliest clinical sign. Any of these ocular nerve paralyses may be complete or incomplete, permanent or transient.

3. *Temperature.*—A certain amount of temperature has been present in all of the cases which I have seen.

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The degree has varied a great deal. In some it has not risen above 99, in others it has never been below 101, and often higher. The rapid types have displayed a high temperature early and persistently. In some the temperature has been irregular and sudden. Sharp rises to 103 or 104 have not been uncommon.

In the slow cases the course of the temperature has been somewhat suggestive of that occurring in typhoid fever, as it has slowly and steadily increased.

4. *A Change in the General State.*—These patients have all appeared sick, they have been difficult to arouse, and when aroused they have lacked interest in their surroundings. They have presented a typical appearance, and as seen from day to day have looked worse. Their facies have been characteristic and their color bad.

The mild cases simulate influenza. They begin slowly and gradually, become more marked and then subside. They may present two or three of the characteristic symptoms, such as fever, cranial nerve paralyses and twitching, or they may simply annoy the patient with slight malaise, double vision and pain. Temperature is not always present in the mild types. Their course is measured by days and their outcome is almost invariably good.

From this slight sketch, it is evident that the symptoms and clinical types of the disease are dependent on the particular part of the nervous system involved. Thus, if the globus pallida is affected, tremor and rigidity of the parkinsonian type will result; if the nuclei of the seventh nerve, facial paralysis; if the meninges, which is rare, rigidity; if the posterior spinal nerve roots, pain in the region involved.

Any part of the nervous system may be involved, cerebral or spinal, usually both. The disease in its clinical aspect suggests cerebrospinal syphilis and multiple sclerosis, so diverse, so variable and so multiple are its manifestations.

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I have seen cases of encephalitis lethargica in which there was both facial paralysis and deafness, others in which there was ptosis and strabismus, others in which the pupils were unequal. At one time the choreiform movements were very general. They simulated those of a Sydenham chorea and were very variable in distribution. A curious manifestation and one which called for a great deal of comment was the appearance of choreiform movements restricted to the abdominal muscles. For some time it was considered that such a sign was pathognomonic, and to it, therefore, was attributed very great importance.

The laboratory did not prove of very great help. The blood examination showed nothing; occasionally leukocytosis. The cerebrospinal fluid was not always uniform. In the great majority of the cases the cell count was increased. In some instances it varied from 30 to 50; in others it ran as high as 100 to 150, or even more. Apparently, an increase, moderate in number, was of better prognostic aid than one either very high or very low.

The urine was of no help.

The ophthalmoscope was of very little help. A very small number of cases have been reported in which there was an optic neuritis; none in which there was a frank choked disk. I mention this because I had occasion to follow carefully one case simulating encephalitis. The presence of a choked disk caused every one consulted to rule out encephalitis lethargica.

The sex and age of the several cases was interesting. Men predominated in the proportion of almost 2 to 1. Whether this was a coincidence or not, I cannot say. The vast majority of these patients were young, strong and robust.

The course of the disease is, except in the mild cases, long, and measured by weeks. It is not at all unusual to have these patients eight or ten weeks in the wards. The cranial nerve paralyses tend to remain and if abating, tend to recur.

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Some of the symptoms merit a descriptive word or two. The facial appearance is very typical. The color is of a grayish, leaden hue, waxlike, the muscles rigid, and fixed so that the face has earned the description, parkinson mask. Ptosis is very common and often bilateral. A patient in a well-marked case of encephalitis will, therefore, present a masklike face with closed eyes. If, in addition to this, the apathy is extreme, the picture will be typical and the diagnosis easy.

Euphoria is frequent. Most of these patients do not complain. Those who are very ill are of course too sick to be cognizant of their condition; but those who are neither delirious nor very stuporous do not seem to feel sick. They rarely complain, the one exception being those affected with radiculitis. It is usual to hear them say that they feel well and that they expect soon to be up and about.

Choreiform movements are common. They resemble those seen in Sydenham's chorea. In distribution they may be general or localized. I have seen patients with general twitching, hemiplegic twitching or jerky movements restricted to one particular part, as the hand, face or abdomen. I saw several cases in which the choreiform movements were localized in the abdominal muscles.

Both sphincters may become involved. Rectal involvement is a late development, and when present renders the prognosis very grave. I saw one case which began with difficulty in urination. This patient was admitted to the surgical side of the hospital for retention, and it was only after two or three days' observation that the correct diagnosis was reached. This is, however, very unusual. A great many of the cases had to be catheterized. I can recall one period when in the wards of the hospital there were three encephalitis patients each of whom had to be catheterized. Catheterization was often necessary early in the course of the disease.

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Photophobia was very general; it was one of the early manifestations occurring in conjunction with the coryza and thereby suggesting measles, an ordinary cold or influenza rather than encephalitis.

The speech is frequently involved. It is difficult to describe, as it is neither paretic nor scanning. It is thick, uncertain and hesitating. There were many instances in which the speech, uncertain and peculiar as it was, became still further modified by the involvement of the facial muscles in choreiform movements. Mental symptoms are very usual in encephalitis. They are most frequent in the severe cases; a severe psychosis, if I may use the term, means a severe encephalitis. The pulse is rapid and becomes more so as the disease progresses. It is not at all unusual to see these patients with a pulse of 120 to 140.

Any of the cranial nerves may be affected except the first. Cases of optic neuritis have been reported, of difficult and slight deafness, etc.

The pathologic changes which encephalitis presents consist of several varieties.

There are congestion, edema, hemorrhage, chromatolysis and infiltrative lesions.

Macroscopically, there is a good deal of edema with marked venous congestion. The convolutions may be somewhat flattened and the vessels of the pia much congested. On section, the basal area is red and very congested. Closer examination of this area will show slight patches of softening and large or small hemorrhagic areas.

Microscopically, there is seen the areas of softening, the areas of hemorrhage, the edema, the degeneration of nerve cells and the infiltration around the blood vessels of small round cells.

The apathy differs from any other form of lethargy. These patients sleep most of the time. They can be roused but only after considerable effort, and at once relapse back into stuporous sleep. It is usual to wake

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them to feed, and in the midst of the feeding to have them lapse back into stupor. They will rouse, answer a question or two, and in the midst of the conversation close the eyes and go back to sleep.

I have mentioned the more usual symptoms, but many others may occur dependent on the particular part of the nervous system involved, and the extent and severity of the infection. Thus one may find Kerwig's sign, rashes, pupillary inequality and pain.

I saw several children suffering from encephalitis. The disease with them appeared to assume a much milder and shorter course than was the case with adults. I saw no old people with encephalitis. The oldest case that I was called to see was 50 years of age. To give you a more definite idea of the age at which this disease occurs I can state that the ages recorded of the nine histories which I have here were as follows: 21, 38, 21, 33, 50, 28, 16, 38, 35.

The cause of the disease is still obscure. Many authorities have reported the isolation of a specific virus. In my service at the City Hospital in New York, the pathologist, Dr. Larkin, found in the brain of an undoubted case a filtrable virus, as did Dr. Strauss at the Mount Sinai. But the virus has not been recoverable in all cases, nor has the monkey in whom the virus has been injected in every case developed paralysis. It is true that in some cases he has. The epidemic was most prevalent a few weeks after the epidemic of grip which occurred last fall and in the early winter. The interesting question which a discussion of encephalitis always brings up is whether or not any connection exists between influenza and encephalitis.

The probable port of entry in this disease is the nose. This is proved by the constancy with which the virus has been discovered in the nasal washings and the frequency with which coryza ushers in the disease. If this is true, and I am strongly inclined to believe that

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it is, the condition is mildly contagious. It ought, therefore, to be safeguarded against and patients with encephalitis ought to be isolated.

Encephalitis lethargica, therefore, is an acute infection due to a specific virus. It seems to have some connection with influenza, as most of the epidemics have followed similar epidemics of influenza. It probably is akin to poliomyelitis, in that it is an inflammation of the gray matter of the cord it is a form of poliomyelitis. It is probably contagious, but only mildly so; otherwise many cases would have developed in the general wards. I knew of none in New York; but as I have already said, I knew of three interns and one nurse.

41 East Sixty-third Street.

ABSTRACT OF DISCUSSION

Dr. Frank R. Fry, St. Louis: The profession generally has not yet become well enough orientated on epidemic encephalitis. Hence papers of this kind are timely. Dr. Hunt has given certain details which help to illuminate the clinical character of these invasions. I have seen only one case in which there were complications of importance outside of the nervous system. The patient had an arthritis affecting most of the joints. In the larger joints it passed away, but the wrists and some of the finger joints were badly deformed. Similar cases have been reported in the literature. In comparing this disease with other infectious processes, I believe I have noted unusually prompt relief by flooding the system as rapidly as possible with water. As to etiology, I was impressed by a statement made by Dr. Peter Bassoe that epidemic encephalitis is due to a virus that at some time and in some way has had connection with influenza.

Dr. Julius Grinker, Chicago: This disease seems to invade almost any part of the nervous system. Recently I saw a typical choked disk in a case of lethargic encephalitis. Choked disk has not so far as I know been recorded, nor does any one seem to have seen a case. I have not heard Dr. Hunt state that a poliomyelitis may accompany the encephalitic process. I called my case a polio-encephalomyelitis lethargica. My patient developed symptoms of encephalitis with ptosis, sleepiness, hypersomnia, and within a week from the onset, there appeared a distinct peripheral facial paralysis. However, the first symptom was a left sided hemiplegia evidencing every phenomenon of a central paraly-

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sis, and this was succeded within a few days by a peripheral facial paralysis on the side of the leg paralysis, and a week later the patient presented all the symptoms of poliomyelitis of the left upper extremity on the same side as the central paralysis. At about the same time there developed double optic neuritis. The patient was recovering when I left home; there was recovery from everything except the atrophy in the left arm and hand muscles, the neuritis having changed into a secondary optic atrophy. In other words, a choked disk and a real poliomyelitis syndrome was added to the encephalitis. I think if anything throws light on the etiology, the poliomyelitis syndrome in this case shows that it belongs to the same family of diseases, depending on the extent of the lesion, whether it shall be a case of encephalitis or an encephalopoliomyelitis.

Dr. Tom A. Williams, Washington, D. C.: Many of the patients I have seen had no lethargy at all. It is only the most severe cases that show lethargy. The pathology attributable to it is based on that of the most severe cases of basal involvement. But throughout the brain are to be found the same pathologic appearances but less intense. It is involvement of the stem medulla structures which causes death in most instances of this disease. However, we should not use the name lethargic encephalitis, but term the disease chidemica acuta as so many physicians think that lethargy means the disease. In one case in Washington the diagnosis of "lethargic" encephalitis was made because the patient passed into the comatose state. When the patient died a frontal neoplasm was found. This patient showed spinal lymphocytosis also. In our epidemic (Washington) of 1910 we had 500 cases, a great many of which became comatose, especially children. Thus the diagnosis of encephalitis can only be surmised in many cases even when you do find a slight degree of lymphocytosis in the spinal fluid. There is a midbrain form, a form with only mental disturbance, and the form with the spinal symptoms most conspicuous. I have seen the Babinski sign present in some cases. It is not always possible to make a diagnosis, except by most careful study of the neurologic aspects of the case, when, with some reserve as to the diagnosis, we can say that any rapid, mild infective process, accompanied by marked neurologic signs, in the absence of syphilis, may be a case of encephalitis acuta.

Dr. Ross Moore, Los Angeles, Calif.: In quite a series of cases in Los Angeles not one patient seen by me had a definite condition of lethargy. It was a pseudolethargy out of which the patient could be brought by a very ordinary question asked in a quiet ordinary tone.

Dr. Climenko, New York: Strauss and Loewe, of Mt. Sinai Hospital, have been able to demonstrate a germ and reproduce it in several generations of monkeys and rabbits.

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They have shown definitely that the portal of entrance of the disease is through the nose. It is useless to consider types in lethargic encephalitis. All sorts of types of the disease are seen. When the first cases of lethargic encephalitis appeared in New York all the best neurologists were baffled. They saw cases of multiple sclerosis develop over night, likewise paralysis agitans and general paresis. Every part of the nervous system can be involved, the central as well as the peripheral system. Many patients gave a history of trifacial neuralgia. One case was suspected to be appendicitis, and another was thought to be an ulcer of the stomach because of a unilateral radiculitis. The youngest patient I have had was a child of 7 weeks, but others have reported even younger children than that. In the wards of Mt. Sinai Hospital I saw a man of 65 affected. The disease is found in both sexes about equally.

Dr. Wilson of London was the first to call attention to the fact that the term encephalitis lethargica is a misnomer. Not all patients are lethargic and not all of them sleep; on the contrary, many patients cannot sleep at all. Insomnia is one of the incipient symptoms.

Economo, who was probably the first to describe this disease, holds that the optic thalamus is responsible for the somnolence. In pituitary disease somnolence is one of the symptoms. Such changes occur in hibernating animals. If the portal of entrance may be the nose, it is easy to conceive that pituitary involvement is responsible for the disease.

In regard to the duration of this disease, I know patients who were discharged from the hospital apparently perfectly well and months after, they developed a new set of symptoms far more severe than the initial symptoms.

Dr. George A. Moleen, Denver: The term encephalitis lethargica is an unfortunate one. It does not describe any of the findings. Probably the term noninflammatory hemorrhagic polio-encephalitis meets the situation both from a pathologic and an anatomic standpoint. A slight digression in the clinical history from those which have been observed which points to a bulbar predominance was noted in the case of a young man who had what was said to be influenza. Apparently he recovered, but about a week later he presented an incoordination which resembled a cerebellar ataxia. A slight diplopia was noted and it was believed to be due to slight weakness of the external rectus muscle of the right eye. There was no lethargy, no delirium, the morning and evening temperature was the same—i. e., no morning remission. There was also a slight weakness of the muscles of the right side of the face, but not so much so on emotional movements. The following day he was unable to swallow, and the palate was immobile with phonation. No reflex movement was obtained from the pharyngeal wall or soft palate. Some days after this he developed definite atrophy of the right side of the

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tongue with atrophic angulation of the tongue. This persisted for about a week. About two days after atrophy of the tongue developed he began to show weakness of the stenocleidomastoids and later of the scaleni. From this time the course of the disease was checked. The temperature curve changed and showed the usual morning and evening variations, and all other symptoms gradually disappeared. The patient showed the typical bulbar symptoms without sleepiness or the mental disturbances described. All microscopic examinations of the spinal fluid and blood were negative.

Dr. Oscar J. Raeder, Boston: I have seen cases in which we thought feeblemindedness was present. In one case the only symptom was sleepiness, a sort of hebephrenia and inability to take interest in or do the usual things. We thought there might be a pituitary involvement. However, after a few months the child gradually began to improve and she has steadily improved. In children this type of encephalitis often gives very mild manifestations.

Dr. Andrew L. Skoog, Kansas City, Mo.: I saw a patient who exhibited peculiar rhythmical purposeless movements in the lower right abdominal quadrant. These movements were a little coarser than we usually see with chorea. They spread rapidly. Within two or three days the upper and lower abdomen and all of the spinal muscles were involved. This patient has presented a marked insomnia. I did a lumbar puncture and found a mild lymphocytosis of the spinal fluid and a little increased pressure. Otherwise the findings were negative. I made a diagnosis of lethargic encephalitis. The patient passed into a state of lethargy. I would suggest the name nona for this disease. It was employed for the earliest reported cases years ago in Italy.

Dr. Roy M. Van Wart, New Orleans: In this section of the country we have had quite a number of cases the outstanding feature of which was absence of lethargy. In a few cases the patient presented some stupor, but lethargy was not the outstanding factor. None of the cases seemed to fall into the classical group described by Dr. Hunt. In two cases the initial symptom was pain in the sciatic nerve; nothing else found. These patients developed symptoms of the disease with paralysis and finally death resulted. In another case active meningeal symptoms developed with very high temperature and terminated fatally. In a child, aged 3 years, meningeal symptoms were the most prominent. In still other cases the symptoms were double facial paralysis with pains in the arms and complete paralysis of both legs, evidently due to myelitis. Lethargy has not been a prominent symptom, and the character of the cases made us extremely doubtful as to what they were. Another case in which we were doubtful as to the diagnosis, choked disk was present.

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This patient recovered. There was no other cause for the choked disk.

Dr. Albert Woldert, Tyler, Texas: Flexner claims that the severity of the cerebral lesions diminishes from before backward; the upper or cervical cord often shows changes; but it is still to be determined how often and to what extent the cord as a whole is affected. This assertion might lead one to suppose that the micro-organism of this disease gains entrance through the nose and throat, as occurs in acute anterior poliomyelitis. The micro-organism which causes encephalitis lethargica probably is of minute size, and filtrable. In the endeavor to discover the nature and mode of conveyance of certain diseases by experiments some investigators have gone about their work in a way somewhat contrary to the way Nature does things, just as I did in my work to determine the mode of conveyance of malarial fever, through the medium of the mosquito in 1899, about a year before the yellow fever commission was appointed. I placed the mosquitoes in large test tubes and afterward endeavored to force them to bite a malaria patient. After I had almost finished my work, Dr. Ronald Ross, at that time of Liverpool, suggested that it might have been better if I had placed mosquitoes under a mosquito bar, and allowed them to bite the patient in a perfectly natural way. It is to be hoped that those who are endeavoring to determine the cause and mode of transmission of lethargic encephalitis will not waste time on methods that are too artificial but will arrange their experiments so as to conform to the natural way.

Dr. David S. Booth, St. Louis: In one case I was undecided regarding the presence of this disease notwithstanding a positive blood Wassermann. I really believe there was also a lethargic encephalitis. Another point is the difficulty of differentiation from serous meningitis.

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THE PHYSIOLOGIC SIGNIFICANCE OF THE BABINSKI TOE RESPONSE

I. LEON MEYERS, M.D.
Attending Neurologist, Cook County Hospital
CHICAGO

The abnormal plantar response that occurs in disease of the pyramidal tract, as described by Babinski,[1] consists of a group of simultaneously occurring movements, the most conspicuous of which are dorsiflexion of the big toe, separation—generally associated with plantar flexion of the other toes and eversion of the foot. The reflexes with which the phenomenon should properly be identified are, therefore, the coordinate or purposive reflexes. These reflexes, in addition to exhibiting themselves in simultaneous contraction of a large number of muscles, are like the Babinski response in that they occur exclusively or are especially prominent after the cerebral influence has been removed. Sherrington[2] and Riddoch[3] give details concerning these reflexes in the "spinal" animal and "spinal" man.

PREVIOUS INTERPRETATIONS OF THE BABINSKI TOE RESPONSE

Among the explanations of the origin and mechanism of the toe response are: Knapp[4] attributed the dorsiflexion of the big toe to a greater permeability of the neuron paths of the extensor longus hallucis. Van Woerkom[5] considered this movement of the big toe a manifestation of a primitive function existing when man, in his evolution, abandoned an arboreal life to lead one of terrestrial progression. Marie and Foix,[6] noting that with extension of the big toe there is a shortening of the stimulated limb, concluded that the reflex

1. Babinski, M. J.: Compt. rend. Soc. de biol., 1897; Semaine méd. **17**: 321, 1898.
2. Sherrington, C. S.: J. Physiol. **5**:28, 1910.
3. Riddoch, George: Brain **40**:264, 1917.
4. Knapp, P. C.: Rev. Neurol. & Psychiat. **5**:825, 1907.
5. Van Woerkom: Rev. Neurol. **22**:285, 1912.
6. Marie and Foix: Rev. Neurol. **23**:657, 1912.

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represents the flexion phase of the step, or of the "mark-time" movements of Freusberg, Sherrington and others. They thus consider the Babinski toe response to be the "minimal motor response" of that phase of the step in which the limb is advancing to a new position.

Walshe,[7] who studied the flexion reflex, flexor spasms and the Babinski toe reflex, maintains that all these phenomena are identical in character. The toe response is thus part of that type of flexion reflex which is purely defensive, such as the withdrawal of the limb on the application of a hurtful stimulus. Whereas Marie and Foix look upon the toe response as the "minimal motor response" in the flexion phase of the step, Walshe considers contraction of the hamstring muscle, which occurs synchronously with the toe response, as the "minimal motor response" of the movement of defense.

Any one of these theories is open to the objection that it does not explain the various movements that accompany dorsiflexion of the big toe, such as the fanning out and plantar flexion of the other toes, eversion of the foot and especially contraction of the quadriceps cruris, an extensor of the leg, and contraction of the gluteus maximus, a pure extensor of the thigh. That there is synchronously with the Babinski toe response contraction of the quadriceps cruris has been noted by Cohn,[8] Jacobsohn and Caro,[9] Erwin Loewy[10] and myself. But I have also noted that there is simultaneously an extension of the thigh on the hip. The contraction of the gluteus maximus producing this movement can be frequently observed when the toe sign is obtained while the subject is in the prone position. This is shown graphically in Figures 4 and 5.

To obtain these graphic records, I employed this method: A piece of cord attached to the big toe passed under a pulley to a writing lever in contact with the drum of a kymograph. Dorsiflexion of the big toe was thus recorded by an upstroke of the lever. To record the other movements in the limb I employed the apparatus shown in Figure 1.

To the middle of a leather braclet (1) is fastened a metal plate (2). To this plate are riveted a semirigid metal strap over 5 inches

7. Walshe, F. M. R.: Brain **37**:269, 1915.
8. Cohn, T.: Neurol. Centralbl. **30**:1107, 1911.
9. Jacobsohn and Caro: Neurol. Centralbl. **31**:420, 1912.
10. Loewy, Erwin: Neurol. Centralbl. **32**:90, 1913.

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long (7) which extends along the surface of the limb in its long axis and a rigid bar (3) rising from its point of attachment so that its end is a couple of inches above the free end of the metal strap. Through the free end of the bar passes a metal rod (4) which rests by a small circular plate (5) on an elastic air chamber (6), fastened to the free end of the metal strap. The rod is secured by a thumb screw where it passes through the bar so that the circular plate is kept firmly in contact with the air chamber. By a rubber tube the air chamber communicates with a Marey's tambour and a kymograph.

To record the contraction of the quadriceps cruris and the hamstring muscle, the bracelet encircled the thigh at the lower end and the air chamber rested, respectively, at about the middle of the quadriceps or on the hamstring muscles just below the gluteal fold. To record the contraction of the gluteus maximus the bracelet encircled the thigh at about its middle and the air chamber rested on the lower part of the muscle which extends between its origin at the sacrum and coccyx and its insertion. I recorded the flexion of the knee on the thigh by having the bracelet encircle the thigh at about its middle and the air chamber rest at the lower end of the popliteal space between the two heads of the gastrocnemius. I have graphic records of the movements in the limbs associated with the Babinski toe response from fourteen hemiplegic or paraplegic patients. The results obtained are shown in Figures 4 to 7. The records show that simultaneously with the Babinski toe response there was invariably a contraction of the homolateral quadriceps cruris, gluteus maximus and hamstring muscles. This response was not accompanied by movement in the contralateral thigh (with the exception of slight contraction of the hamstring muscles) when the stimulus was mild. When, however, the stimulus was strong, the contraction of the quadriceps was immediately followed by its relaxation and this was accompanied by contraction of the contralateral quadriceps. In the one case of Friedreich's ataxia I have obtained results in some respects, directly the reverse of the preceding. The records showed that simultaneously with the Babinski toe response there was flexion of the knee with relaxation of the homolateral quadriceps and gluteus maximus (Figs. 11 and 12). This may have been due to the muscular atonia in this disease. In contrast to the response on the paretic side in hemiplegics, was that obtained from the normal limb. When the stimulus

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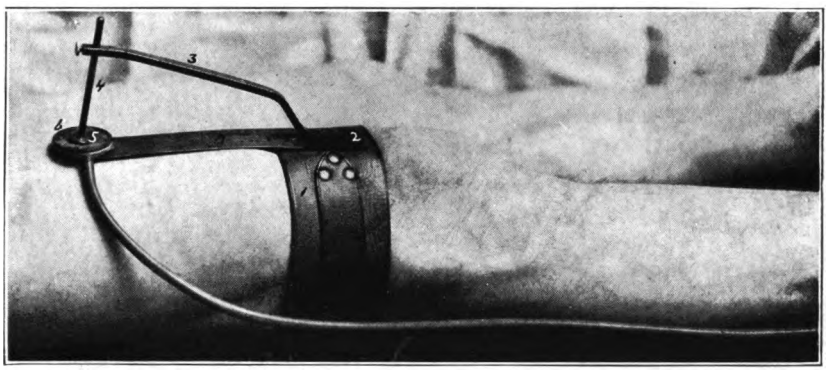

Fig. 1.—Apparatus for recording the movements in the segments of the limbs.

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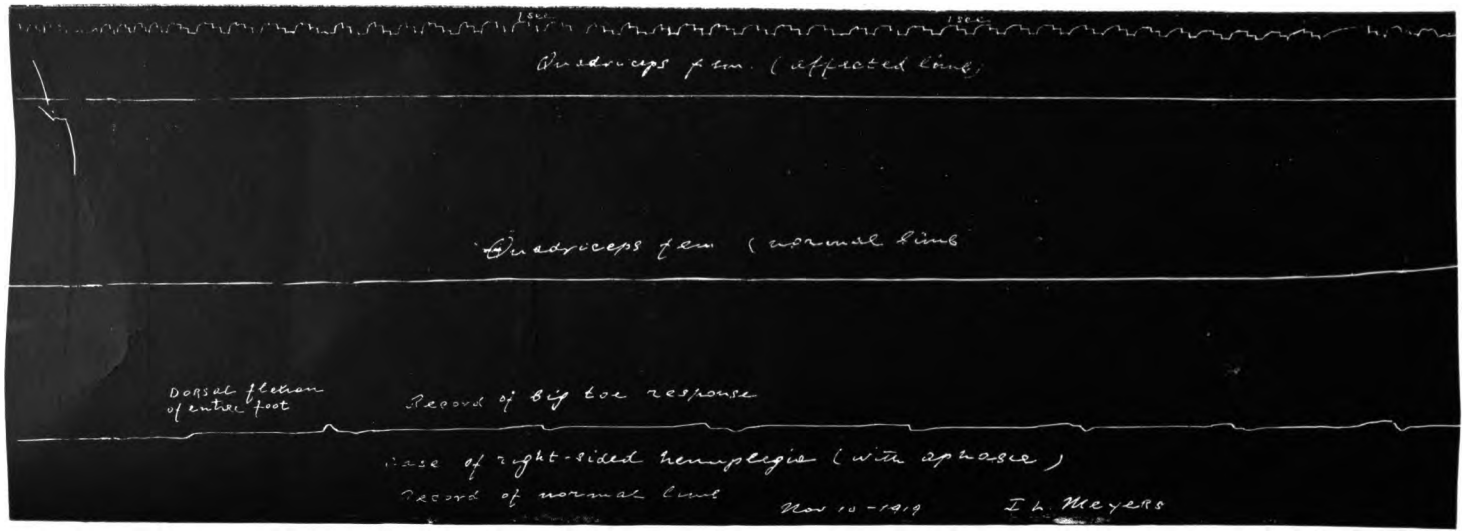

Fig. 2.—Record of the normal plantar response.

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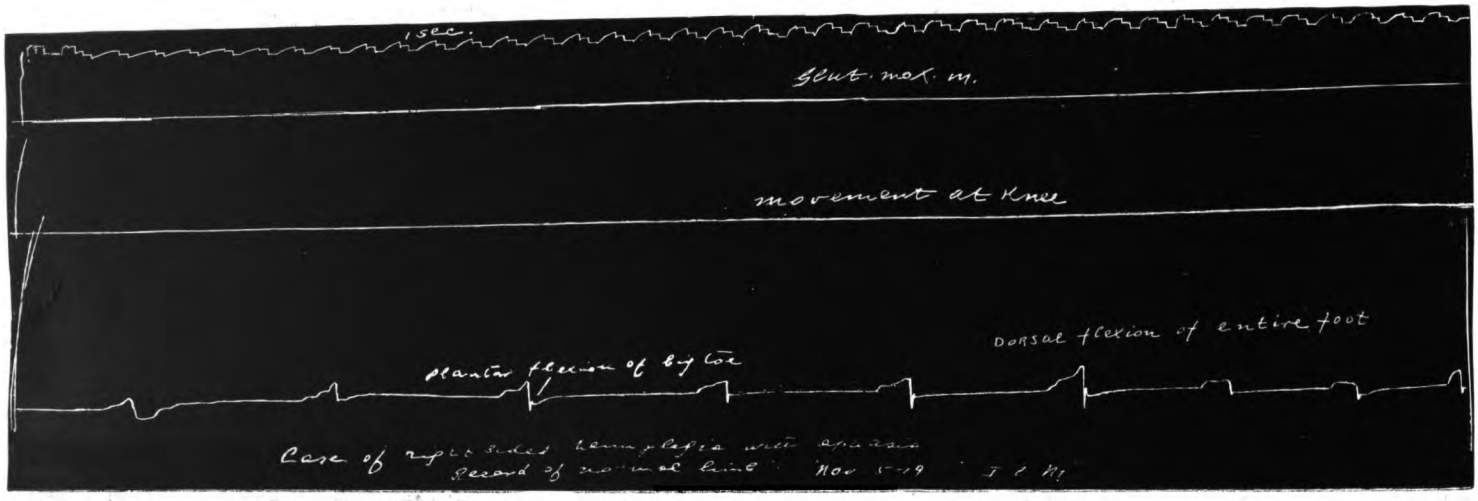

Fig. 3.—Record of the normal plantar response.

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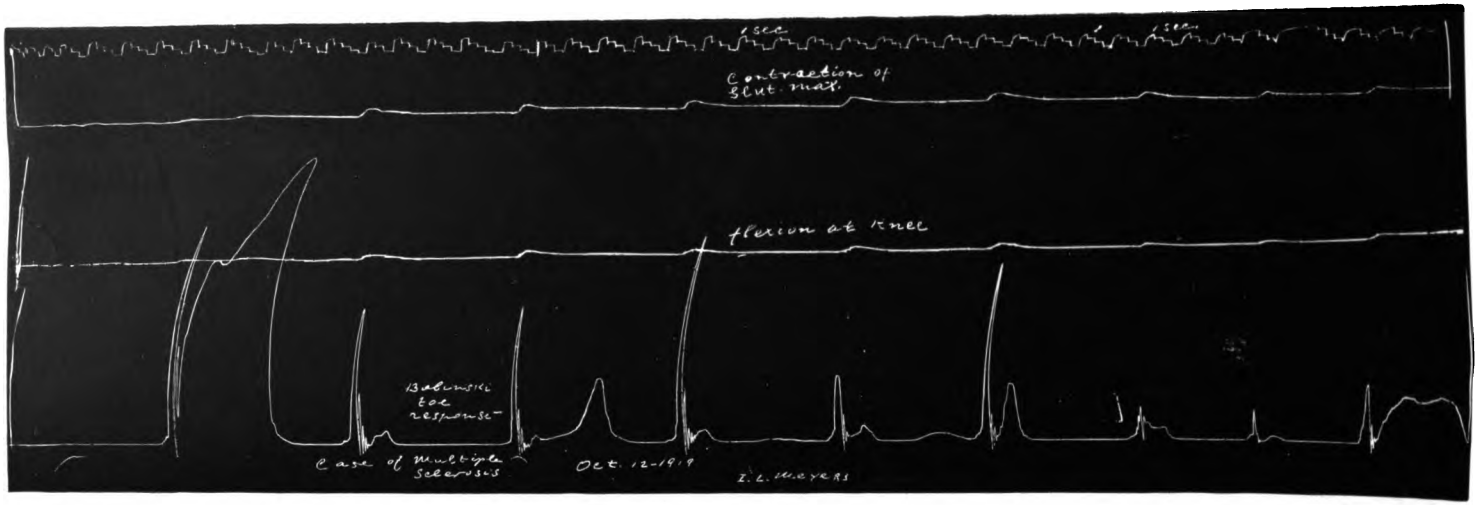

Fig. 4.—The Babinski toe response with the accompanying movements in the other segments of the limb.

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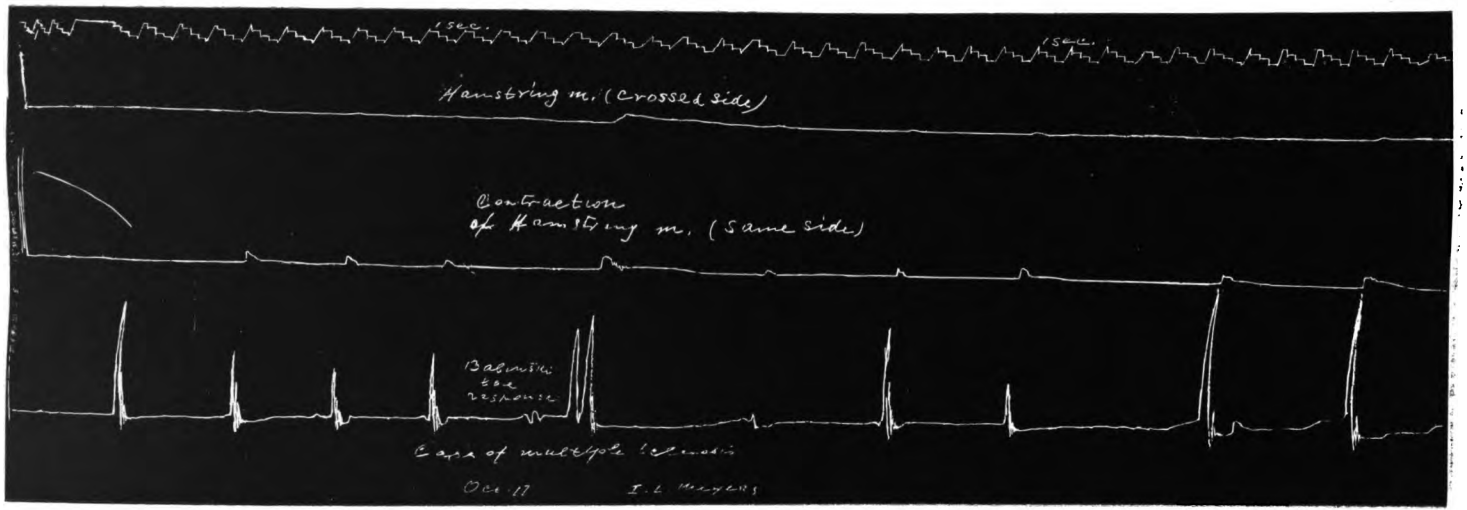

Fig. 5.—The Babinski toe response with the accompanying movements in the other segments of the limb.

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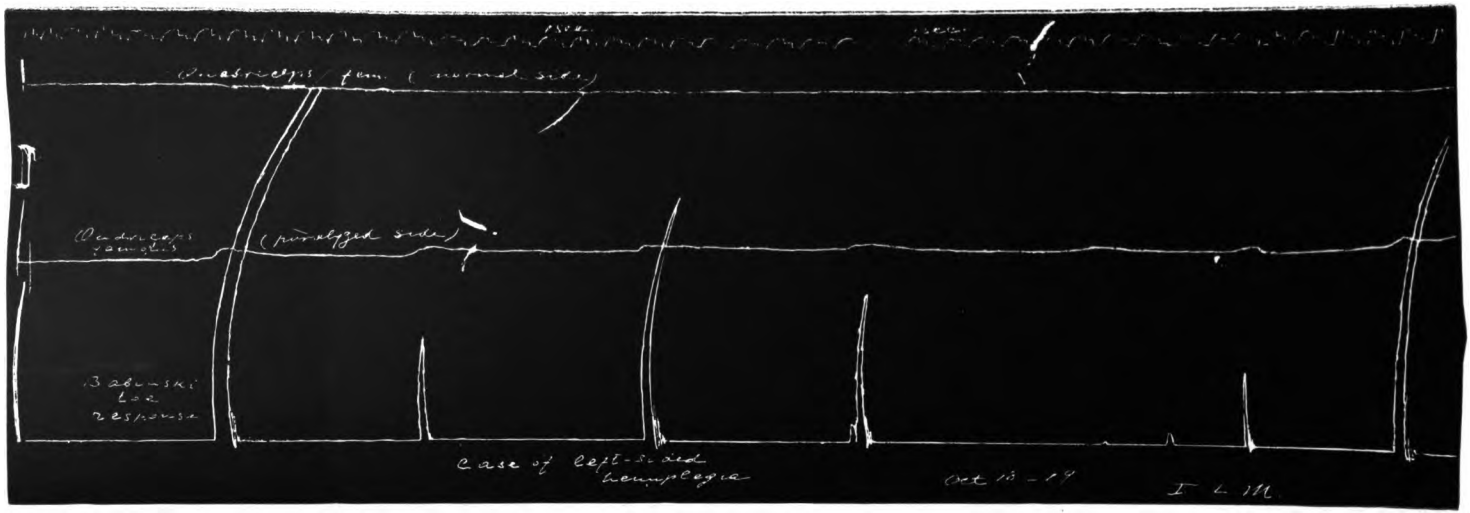

Fig. 6.—The Babinski toe response with the accompanying movements in the other segments of the limb.

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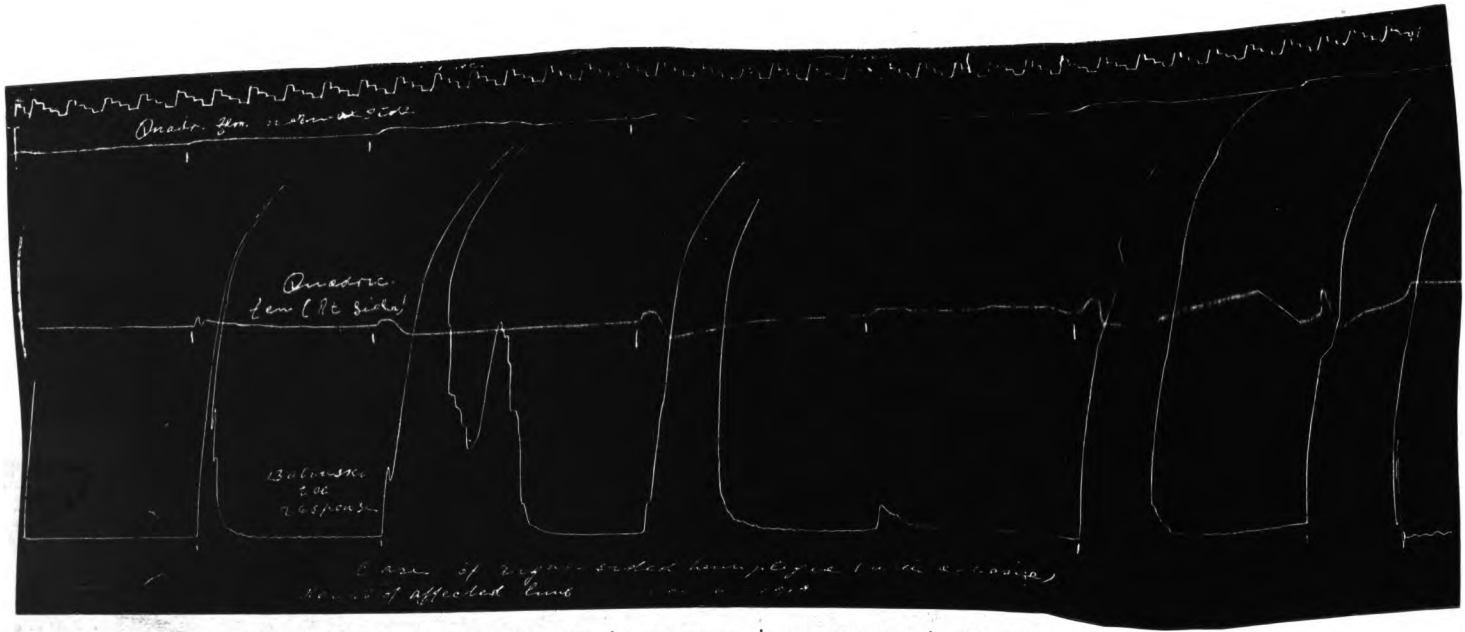

Fig. 7.—The Babinski toe response with the accompanying movements in the other segments of the limb.

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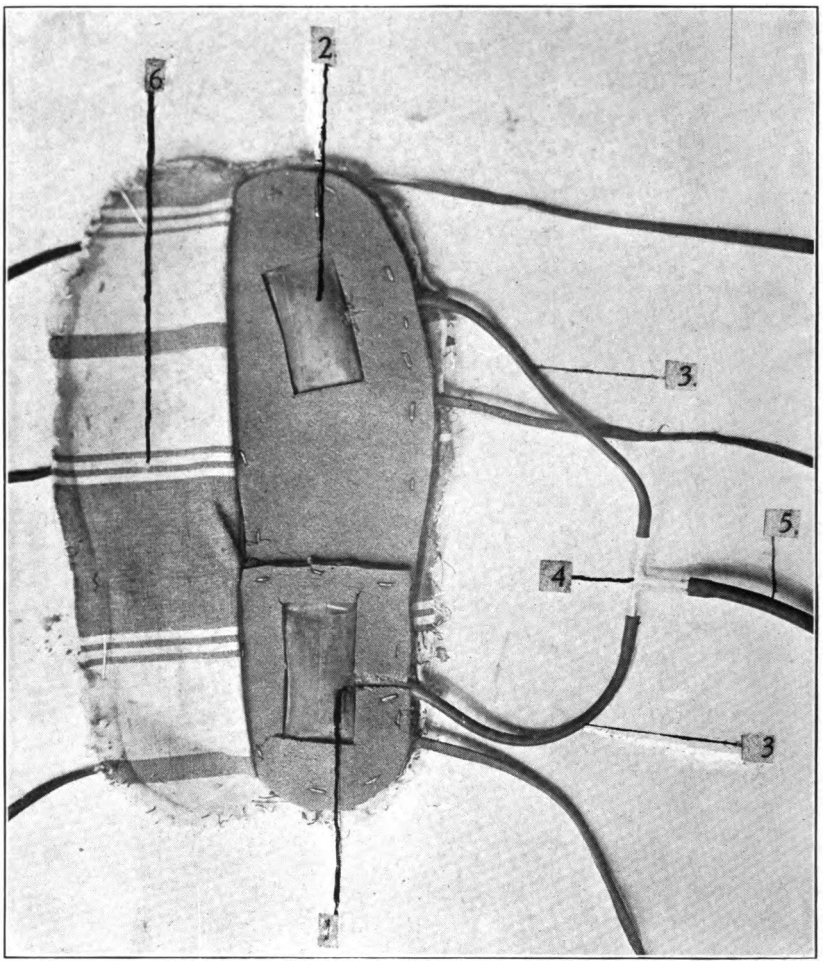

Fig. 8.—Apparatus for recording the movements of the foot in walking.

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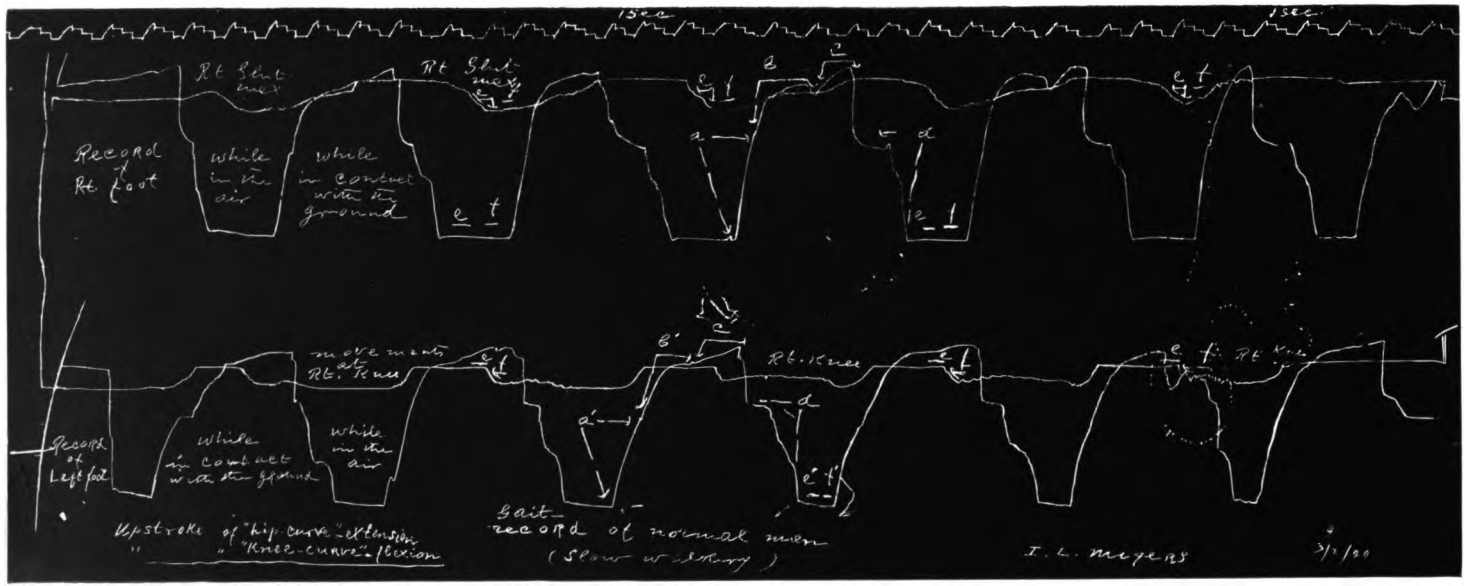

Fig. 9.—Record of the various phases of the step.

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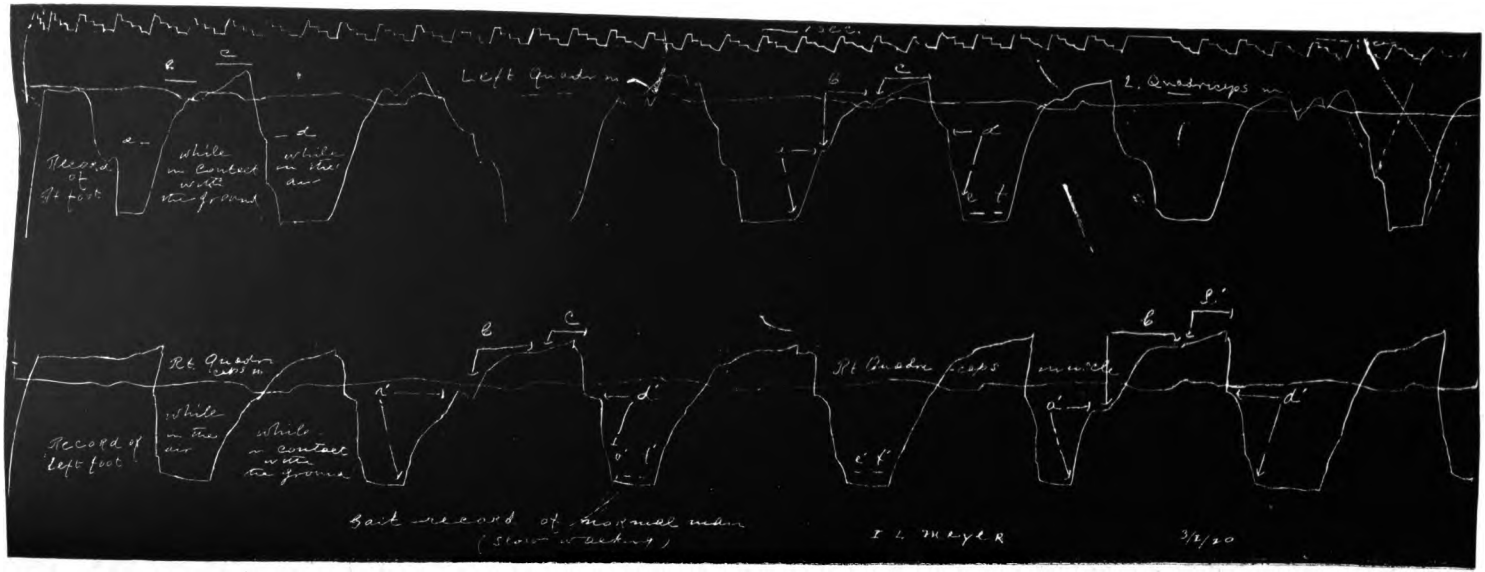

Fig. 10.—Record of the various phases of the step.

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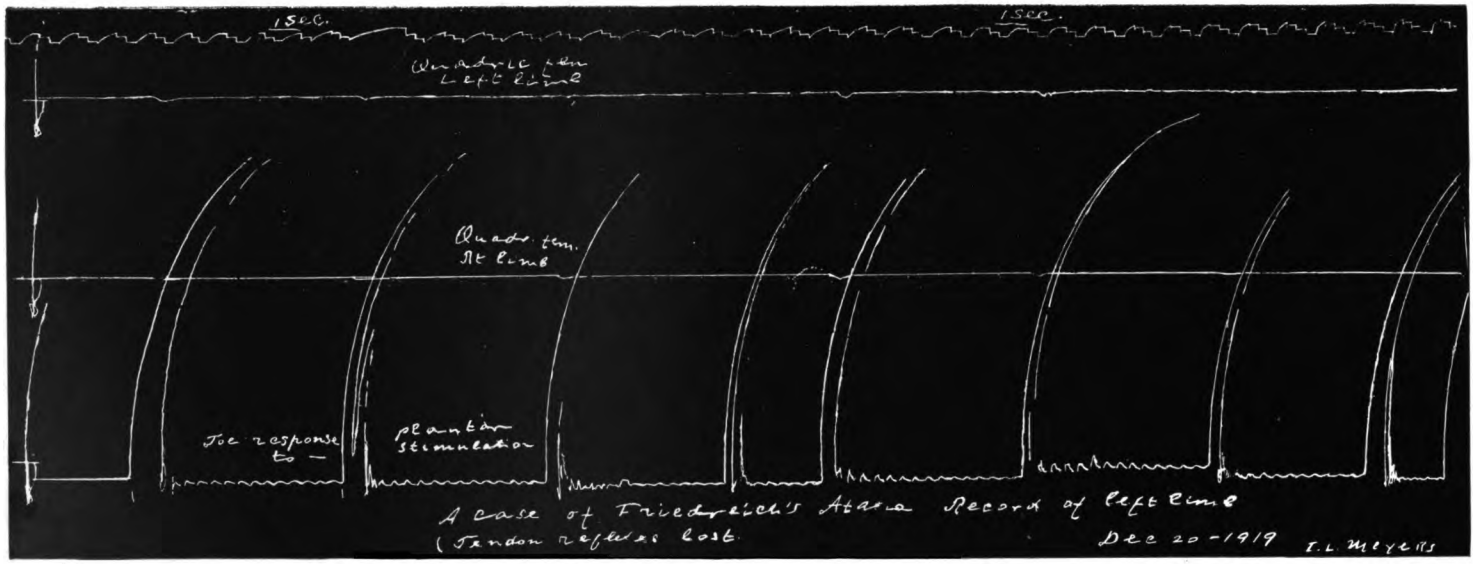

Fig. 11.—The Babinski toe response in a case of Friedreich's ataxia.

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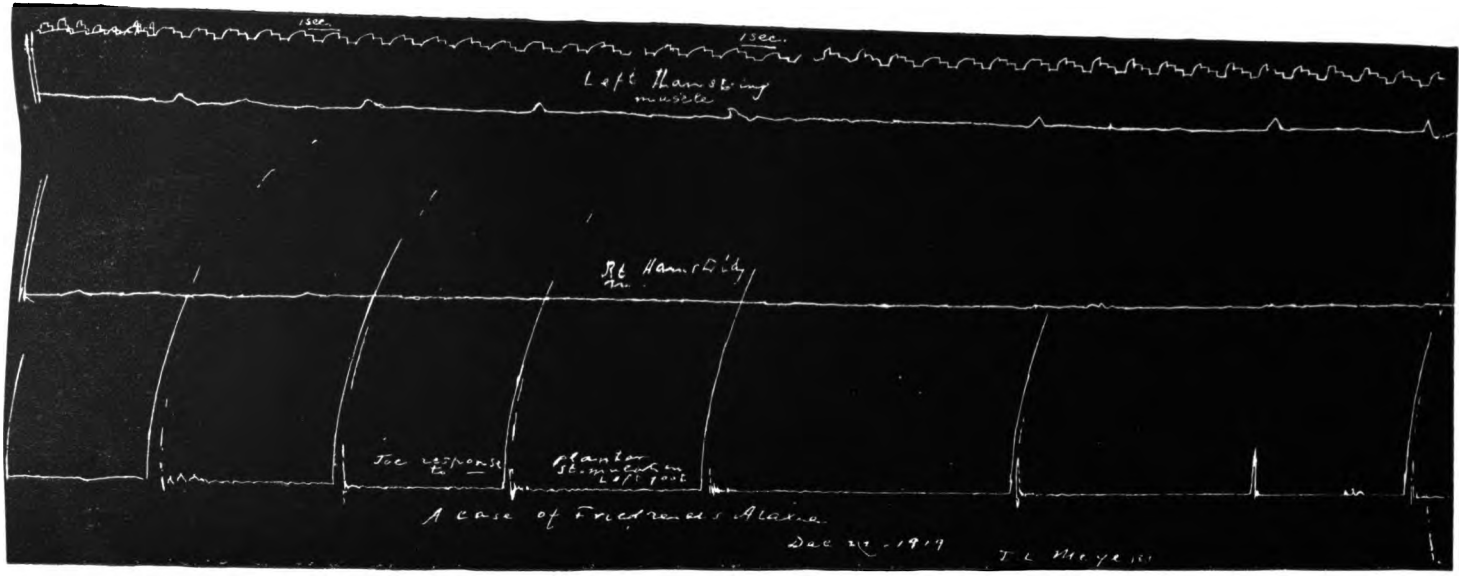

Fig. 12.--The Babinski toe response in a case of Friedreich's ataxia.

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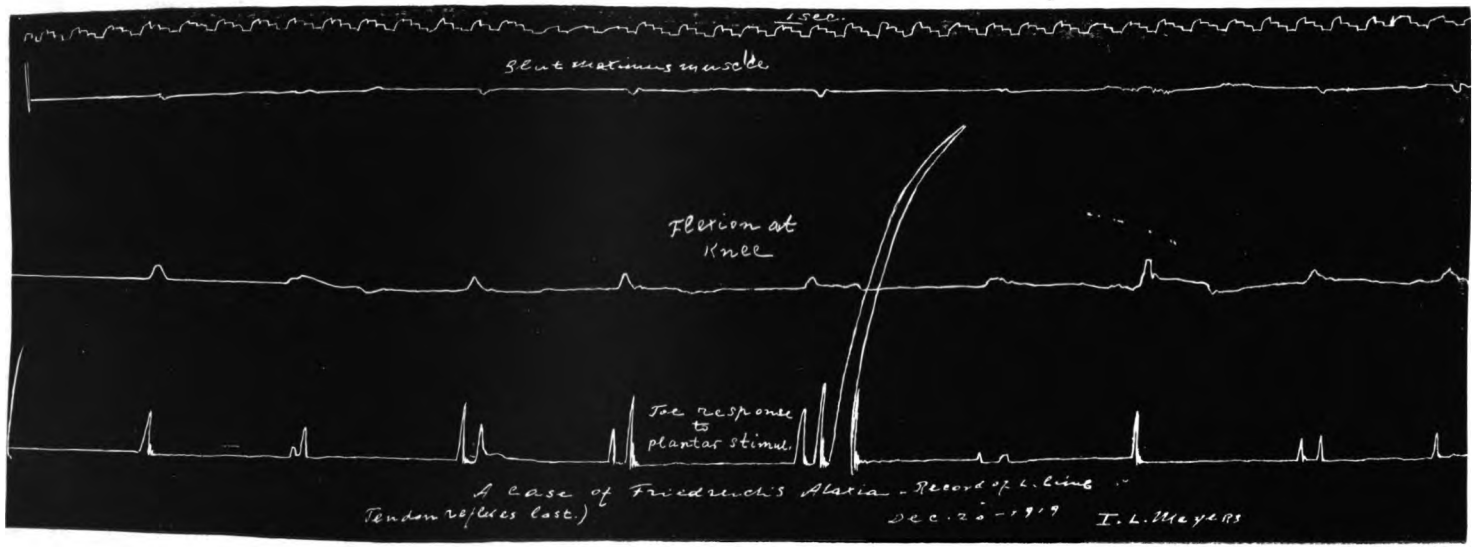

Fig. 13.—The Babinski toe response in a case of Friedreich's ataxia.

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was strong the limb underwent a rapid movement of withdrawal, entirely too rapid to be properly recorded; when the stimulus was mild, there was dorsiflexion of the foot, generally immediately followed by plantar flexion of the big toe (the only toe the movements of which were recorded), with no movement in the other segments of the limb (Figs. 2 and 3).

MOVEMENTS OF THE LIMBS IN LOCOMOTION

Since locomotion is the primary and essential function of the lower limbs, I wished to determine whether this combination of movements occurring synchronously with the Babinski sign is reproduced by the limbs at any time during walking. First I considered that the inner metatarsal, as is shown by a number of anatomic facts,[11] forms the medium through which pressure is exerted on the ground during the propulsion period of the step. The outer metatarsus, with its practically immobile articulations, serves chiefly as a guide and point of support for the body and limb while they are rotating anteriorly during this period of the step. Accordingly I fastened to each foot of a normal person a pneumatic pad, such as I employed in my experiments on the locomotor disturbances in disease of the cerebellum (Fig. 8). The anterior air chamber was placed under the inner metatarsal head instead of the center of the ball of the foot as in my previous studies. Records were thus obtained of the movements of the foot while in contact with the ground, as well as of the length of time it is in the air. Movements of the other segments of the limbs during each phase of the step were recorded at the same time by means of the apparatus employed for recording the movements that accompany the Babinski sign. The records obtained are shown in Figures 9 and 10. They show: (1) *a*, the upstroke which initiates the high curve of the record. This corresponds to the moment when the foot is deposited on the ground, coming in contact with it by the heel (Pettigrew[13]). The resulting com-

11. Frazer, J. Ernest: Anatomy of the Human Skeleton, London, J. A. Churchill, 1914.

12. Meyers, I. Leon: Locomotor Disturbances in Disease of the Cerebellum, Arch. Neurol. & Psychiat. **2**:376 (Oct.) 1919.

13. Pettigrew, J. Bell: Animal Locomotion, New York, Appleton & Co., 1874, pp. 8 and 9, 56-59; Design in Nature, New York, Longmans, Green & Co. **3**:1077 and 1122, 1908.

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pression of the air bulb at the heel causes the lever to become elevated. The associated movements in the other segment of the limbs during this phase of the step are: The hip is slightly extending, the knee is slightly flexing, the quadriceps cruris is contracting and the quadriceps of the contralateral limb, which is at this moment exerting pressure on the ground by the toes, (phase *d'*) is relaxing. (2) Immediately following *a*, phase *b* sets in, which is but a continuation of *a*. During this phase (*b*) the whole foot comes in contact with the ground, especially at the heel. The associated movements in the other segments of the limb during this phase are the same as in *a*, except that the extension at the hip is here greatly intensified. The position of the limb is now to a minor degree, similar to that assumed by the limbs at the beginning of a jump, when in order to exert great and rapid pressure on the ground, they are considerably flexed at the knees. The contraction of the quadriceps during these phases, while the knee is slightly flexing (action of the hamstring muscles) is in accord with the view of Winslow,[14] Hunter,[14] Duchenne[15] and others that in movements that are to be accurate and precise, the antagonists act simultaneously with the protagonists. (3) Phase *c*, the pressure on the ground, already at play in the preceding phase, becomes intensified, being now, according to the estimate by Carlet,[16] one-fifth in excess of the body weight. The pressure is now at the ball of the foot exclusively, and in my experiments, at the sesamoid bones of the inner metatarsal, where the air-bulb for the ball of the foot was placed. The associated movements in the other segments of the limbs during this phase are: The homolateral hip is markedly extending, the knee is also markedly extending, the quadriceps cruris is contracting and the quadriceps of the contralateral limb, which is at this time returning to the ground (phase *f'*), is also contracting. (4) Phase *d*, which represents the period when the limb exerts pressure on the ground by the toes only. The pressure on the ground at the toes causes some compression of the air-bulb at the ball of the foot, and is thus recorded by a partial elevation in the curve of the record. During this phase the homolateral hip is markedly extended, the knee

14. Winslow, J. B.: Quoted by C. E. Beevor: The Croonian Lectures, London, Adlard and Son, 1904, p. 52.

15. Duchenne, G. B.: Physiologie des Mouvements, 1867, p. 766.

16. Carlet, G.: Ann. d. sc. naturelles **16**:5, 1872.

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is markedly flexed, the quadriceps is relaxed and the quadriceps in the contralateral limb, at this time in phases *a'* and *b'*, is contracted. *d* terminates the period during which the foot is in contact with the ground, and is followed by (5) *e*, which corresponds to the time during which the foot is in the air and advancing. The movements in the other segments of the limbs during phase *e* are: The hip is flexing, the knee continues to be in flexion, the quadriceps is relaxing and the quadriceps of the contralateral limb, at this time in phase *b'*, is contracting. (6) *e* is followed by phase *f*, when the limb is returning to the ground. During this phase the homolateral hip is slightly extending, the knee is extending, the quadriceps is contracting and the quadriceps of the other limb, at this time in phase *c'*, and exerting pressure on the ground by the ball of the foot, is also contracting. It will be seen thus that a complete step is composed of six different and distinct phases, four of these phases forming the propulsion period of the step and two more the period when the foot is in the air.

On looking now at the phases of the step for the combination of movements, which is observed to occur synchronously with the Babinski sign, we find that it is reproduced in phases *a* and *b*, when the body is exerting pressure on the ground mainly at the heel. During this phase of the step, the following movements take place: The big toe becomes dorsiflexed on the foot; the other toes, as shown by Pettigrew,[18] spread out and generally undergo plantar flexion; the whole foot from the direction of movement of the body and limb, which is not only forward, but as shown by Pettigrew and Morey, also horizontally outward, becomes everted; the knee is slightly flexed; the quadriceps cruris and the gluteus maximus are in contraction, and the quadriceps of the contralateral limb, at this time exerting pressure on the ground by the toes (phase *d'*), is relaxed.

THE PHYSIOLOGIC SIGNIFICANCE OF THE BABINSKI TOE RESPONSE

The following theory thus suggests itself. It may be assumed (Marie and Foix) that the function of progression is represented in the spinal cord (l' automotisme medullaire); that normally the movements of progression are kept more or less in check by inhibitory influences of the cerebrum. When, however, inhibition from the cerebrum is cut off as a result of disease of the corticospinal tracts, the spinal centers of progression reassert themselves whenever a stimulus

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reaches them from the limbs, and the stimulated limb or both limbs execute the stepping reflex in a minor degree. The stepping reflex exhibits itself in the Babinski toe response which represents phase *b*, the first and most important phase in the propulsion period of the step. Phase *b* is the only manifestation of the reflex when the stimulus is mild. When the stimulus is intense, the stepping reflex reaches a much wider extent and passes through, to a minor degree, also phase *d*. The quadriceps cruris of this limb, in contraction in phase *b*, thus becomes relaxed, and there is usually at the same time a movement of plantar flexion of the big toe following the original dorsiflexion. A pronounced reflex of this type affects also the contralateral limb, and, while the stimulated limb is in phase *d*, the quadriceps of the former limb contracts. The most striking features of the Babinski toe response are, the dorsiflexion of the big toe, the spreading out of the lesser toes and the eversion of the foot. This is probably due to the close proximity of the muscles involved in these movements to the point of application of the stimulus (the plantar surface of the foot). If, however, the reflexogenous zone (of the Babinski toe response) extends over the entire limb, (and occasionally also the lower part of the abdomen) the stimulus when applied to the upper part of the limb may, as noted by Marie and Foix, produce extension of the limb (marked action of the gluteus maximus and the quadriceps cruris) and no dorsiflexion of the big toe. The effect of the stimulus is in this instance, too, produced chiefly on the muscles close to the point of stimulus and not on the parts distal to it.

This theory of the Babinski sign explains also the so-called crossed plantar response, frequently observed in paraplegia and occasionally in hemiplegia, which consists of plantar flexion of all the toes with extension of the foot. The phenomenon has been studied and interpreted in various ways by Byron Bramwell,[17] Knapp,[4] Fairbanks[18] and others. If the Babinski toe response represents the propelling period of the step that occurs in phase *b*, the contralateral limb at this time being in phase *d'*, the effect of the stimulus to one limb, when the irritability of the nervous system is so great that the reflex affects the other limb also, must be that of plantar flexion of all the toes in the latter limb, the foot being at the same time fully extended at the

17. Bramwell, Byron: Rev. Neurol. & Psychiat. **1**:795, 1903; ibid. **9**:49, 1911.
18. Fairbanks, A. W.: Rev. Neurol. & Psychiat. **9**:225, 1911.

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ankle. Frequent absence of the crossed plantar response may be accounted for by the remoteness of the toes in the nonstimulated limb from the point of application of the stimulus.

This theory may also explain why the Babinski toe response, while obtainable in some conditions by a stimulus applied to any part of the limb and even the lower part of the abdomen, is obtained with such marked facility by pressure at certain foci in the foot and leg. These "receptive foci" are, I believe, the heel, the ball of the foot and the area under the outer malleolus. It will be noted that pressure at the heel and the ball of the foot reproduces the condition which is associated with dorsiflexion of the big toe in walking, and that pressure under the outer malleolus produces an effect on the peroneus longus, the existence of which is, as stated by Huxley,[19] absolutely distinctive of the human foot, a muscle that is essentially an organ of locomotion.

25 East Washington Street.

19. Huxley, T. H.: Man's Place in Nature, New York. O. Appleton & Co., 1896, p. 124.

ABSTRACT OF DISCUSSION

Dr. Andrew L. Skoog, Kansas City, Mo.: It has always been an interesting problem to consider just exactly what the nature of the mechanism of the affair may be. I have never been convinced as to this. I asked Professor Babinski to explain this matter and he did not respond positively to my question. The work of Marie and Foix is very interesting as throwing additional light on this problem. We should look on this as a phenomenon, as has been indicated by all the workers in the Paris school of pathology and physiology. We might consider this a complexity of reflexes.

Dr. Tom A. Williams, Washington, D. C.: How does your theory account for the flexion, say, of the great toe when you stimulate the right foot? As far as I could gather, Dr. Meyers' interpretation would necessitate stimulating the left foot in order to make the right foot flex, because, as I understood him, he seemed to infer that the flexion phenomenon was a synchronous performance—a unilateral synchronous response to the pressure of the contralateral foot on the ground, during which time the great toe of the contralateral foot was in flexion. I could not follow his argument at all and I hope he will make it more clear.

Dr. Gustave Roussy, Paris, France: In France varied opinions are held as to the interpretation to be given the Babinski reflex. It is considered more and more part and parcel of the reflexes of defense. That is the opinion of Marie

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[illegible] of these from the limbs, and the stimulated [illegible] [illegible] the stepping reflex to a minor degree. [illegible] [illegible] and to the Babinski the response which [illegible] [illegible] most important phase in the propul[illegible] [illegible] Babinski for the only manifestation of the reflex [illegible] [illegible] When the stimulus is intense, the steppin[illegible] [illegible] [illegible] [

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19. Huxley, T. H.:
1896, p. 124.

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and Foix, and also of L'Henriette, and it is my own. I think that the extension of the great toe is only the liberation of one of the cutaneous reflexes of defense, since the investigations of Sherrington, Pierre Marie and Foix have shown that it is one of the easiest to liberate, and we see it appear or disappear first in the series of reflexes of medullary origin and often it exists alone.

Dr. M. A. Bliss, St. Louis: Dr. Chaddock has repeatedly demonstrated in the City Hospital at St. Louis that the submalleolar sign and the wrist sign are constantly present in hemiplegics on the contralateral side.

Dr. A. C. Ivy, Chicago: I would like to present a little comparative physiologic evidence in favor of Dr. Meyers' work. By experiments of stimulation it has been established that the cerebral cortex of the pigeon does not contain a motor area, and in a decerebrated pigeon, locomotion is not disturbed. Such a pigeon, however, will show a positive Babinski. This can be demonstrated either in a partially or a completely decerebrate pigeon. Just how these observations are related to the Babinski reflex as it occurs in the human being, who has a motor cortex, I cannot say. But I do believe this is comparative evidence in favor of Dr. Meyers' theory that the Babinski is a reflex associated with locomotion.

Dr. I. Leon Meyers, Chicago: Dr. Ivy and I have demonstrated the reflex he described. It was observed that when we stimulated the foot of a normal pigeon the toes underwent flexion just as they do in grasping the rod on which the bird is to perch, whereas in a decerebrated pigeon the toes spread out in the same manner as they do in the foot that is on the ground in walking. In the crossed plantar response, there is a flexion of the toes in the contralateral, paralyzed foot and no response—in the case of hemiplegia—in the normal foot because the former is the only one that is capable of exhibiting a locomotor reflex, it being released from cerebral inhibition, the normal limb, on the other hand, being still subject to such inhibition. The Babinski response according to my theory, represents the propelling movement of the limb that is on the ground during the execution of the step. The French and English schools of neurology have assumed that the response is a movement of pure flexion. My experiments on locomotion show that during the propelling stage of the step, while there is, broadly speaking, extension of the limb, there is at the same time flexion at the knee, and it is this flexion that occurs simultaneously with the extension of the big toe. This is essentially the difference between my point of view and the theories that have been presented before.

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